DENTAL SURGERY & PATHOLOGY

EIGHTH EDITION

IN this, the eighth edition of this book, new sections have been added and others re-written and re-illustrated, there are many additions in subject matter and illustrations, and the text has been revised throughout and brought up to date. Advances in knowledge concerning variations in structure, regressive changes in the tooth pulp, the pathology of neoplasms, treatment of fractures, accelerated eruption, and pigmentations of the gums and oral mucous membrane are some of the additions which have been made since the publication of the last edition.

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EVELYN SPRAWSON

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DENTAL SURGERY AND PATHOLOGY

DENTAL SURGERY AND PATHOLOGY

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PREFACE TO THE EIGHTH EDITION

Although it is a comparatively short time since the publication of the last edition of this work, additions have accrued to many aspects of knowledge concerning the practice of dental surgery, and though some, at first sight, may appear to be of academic rather than practical interest, all add to the sum of knowledge of matters concerning the well-being of the individual and what may be learned from his dental condition, and tend to a more accurate diagnosis of his physical status from both the medical and dental aspects, which is the true aim of all clinical and other examination. Such additional knowledge has therefore been incorporated in this edition.

Some twenty-five new illustrations have been added, partly to replace old ones and partly to add to the more ready appreciation by the student of the condition under discussion by conveying a visual as well as a mental picture ; additions have been made to the knowledge of variations in the structure of the teeth, and a new section dealing with conditions accelerating the eruption of the teeth included. Though the principles governing the treatment of fractures of the jaws, and indeed the treatment itself, remain fundamentally unchanged, improvements, refinements, and alternatives in the methods of treatment have been introduced ; but the more radical changes, chiefly of surgical innovations, are at this time of war still in the state of trial and experiment, and at this stage may not—from the point of view of the student—be profitably discussed. The section on regressive changes in the tooth pulp has been largely rewritten and reillustrated.

The odontomata, though the chapters dealing with them are but little altered, are brought more strictly into conformity with surgical and, more particularly, pathological teaching and nomenclature, so that only those productions known to be of neoplastic origin retain that name among the many other abnormal productions originating from the dental formative organs. A large number of other additions, alterations and advances have also been incorporated in the text, particularly perhaps in the chapter on tumours of the jaws.

For figures and blocks we are indebted to :—

The Council of the Royal Society of Medicine ; the Proprietors of the *Lancet* ; the British Medical Association ; the National

Dental Association; the American Medical Association; the Publishing Committee of the *British Dental Journal*; the Publishing Committee of the American Dental Association; the Publishing Committee of the *American Journal of Orthodontics and Oral Surgery*; the Medical Committee of the Royal Dental Hospital; The Amalgamated Dental Company, Ltd.; The Cleveland Dental Manufacturing Co.; The Dental Manufacturing Co., Ltd.; the Canadian Dental Research Foundation; Messrs. S. S. White and Co.; Mr. Henry Kimpton; Messrs. Edward Arnold and Co.; Messrs. H. K. Lewis and Co., Ltd.; Messrs. J. and A. Churchill; Messrs. P. Blakiston's Son and Co.; Messrs. Baillière, Tindall and Cox; Messrs. J. B. Lippincott Company; Messrs. Hodder and Stoughton, Ltd.; Messrs. Cassell and Co., Ltd.; Messrs. Mayer and Phelps; Mr. John Murray; and the Oxford University Press.

We have to thank many friends for much valuable assistance: the late Mr. F. St. J. Steadman for the chapter on 'Local Anæsthesia in Dental Surgery'; the late Dr. Stanley Colyer who by particular request undertook the chapter on Radiology, and in the more technical parts had the valuable assistance of Dr. Charles Colyer; Mr. Martin Rushton for the chapter on 'The Remote Effects of Disease about the Teeth'; Mr. S. G. Allen for the chapter on 'Radium and the Dental Surgeon'; Dr. W. A. M. Smart for the sections dealing with the Physiology of Saliva; Mr. Harold Chapman for contributing a section on 'Fixed Appliances in Orthodontic Work' and figures to illustrate orthodontic appliances, and the occlusal classification of abnormalities in the position of the teeth; Sir George Still for revising a part dealing with Pathological Dentition; Dr. J. H. T. Challis for a contribution on 'The Use of General Anæsthetics for the Extraction of Teeth'; Dr. Reginald T. Brain for details of 'Affections of the Skin arising from Reflex Irritation from the Teeth'; Mr. Claude Colyer for a contribution on 'The Radiographic Diagnosis of Caries'; Mr. Arthur Bulleid for a contribution on 'The Bacteriology of Parodontal Disease'; Dr. W. Russell Brain for a revision of the section on 'Neuralgia'; Dr. W. W. Woods, of the Bernhard Baron Institute of Pathology, for invaluable aid in pathological detail; and many others whose names occur throughout the text.

We wish also to tender our thanks to Messrs. Spottiswoode, Ballantyne & Co., Ltd., who have so efficiently dealt with the difficulty in introducing considerable new matter without altering the paging of the last edition, a difficulty occasioned by the shortage of paper and labour created by the war, and whose efficient rendering of proof sheets has very considerably lessened our labours ; and lastly to acknowledge the very valuable help and advice we have so unfailingly met with at the hands of every member of the firm of Messrs. Longmans, Green & Co. Ltd., the publishers of this work, with whom we have come in contact in the course of its preparation.

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DENTAL SURGERY AND PATHOLOGY

CHAPTER I

Pathological Dentition.

Disorders associated with the Process of Teething—Accelerated Eruption — Retarded Eruption — Incomplete Eruption — Impeded Eruption—Failure of Eruption.

(A) DISORDERS ASSOCIATED WITH THE PROCESS OF TEETHING.

(1) GENERAL.

CERTAIN general disorders frequently occur about the time of teething, and considerable diversity of opinion exists as to the exact relationship between these disorders and the process of teething. Formerly, illnesses at this period appear to have been readily regarded as a natural sequence to the process of dentition, whereas the tendency now seems to be in the opposite direction, and in the search for the cause of general disorders dentition is often ignored.[1]

In an endeavour to arrive at the actual facts of the case it is well to remember that weakly children, especially if they have been hand-fed, are prone to general disturbances, and that local trouble in the mouth probably tends to aggravate such conditions. Further, general disturbances occurring during teething are often wholly traceable to other causes; for example, gastro-intestinal troubles may be due to defective feeding, convulsions to a similar cause, and respiratory affections and otitis media to the presence of adenoids. But after due allowance has been made for all known causes there still remain cases of general disturbance which are coincident with dentition, and which cannot be accounted for if they are not to be attributed to the dental condition. There is naturally extreme difficulty in placing dentition in causal relationship with such disorders, because we have to rely mainly on clinical observation; but it seems only reasonable to assume that such a relationship does exist when the occurrence of a disorder not only coincides with dentition but has a special tendency to coincide repeatedly with the eruption of successive teeth. (See also Chapter XXVI.)

---

[1] In compiling this section we have been greatly assisted by the late Sir Frederick Still.

In referring to this question, Still expresses the opinion that there is too great a tendency to assume that because dentition is a physiological process it is incapable of causing disturbance of health. He points out that pregnancy is also a physiological process, and no one would deny that pregnancy may disturb the health in many ways. Still states that, with increasing experience, he is inclined to enlarge rather than to restrict his conception of the *rôle* which dentition plays in the production of disturbances of various kinds during infancy.

Clinical observation clearly establishes the fact that irritation often exists in the mouth during the period of eruption. In most cases the irritation is due to direct pressure of the tooth upon the superincumbent tissue. J. W. White points out that the aperture at the end of the growing tooth is large, and undue resistance of the gum tissue would lead to pressure on the nerve trunks entering the pulp. White supports his view by clinical evidence afforded by cases in which there are no local signs of inflammation and in which constitutional disturbance disappears when the gum is lanced.

The following general disorders may, in Still's opinion, *be traceable to dentition*:—

(*a*) Loss of appetite.

(*b*) Failure to gain weight or diminution in the rate of gain. This occurrence is frequently associated with dentition, sometimes with loss of appetite, but sometimes without. Its explanation is not obvious. The deficiency in weight is usually made up in the intervals between the eruption of the different teeth.

(*c*) Sleeplessness and startings during sleep.

(*d*) Instability of the nervous system. Infants troubled with coming teeth often show a tendency to convulsive twitchings, upward rolling of the eyes, slight strabismus, although not losing consciousness. In children of neuropathic heredity, or who, through rickets, are predisposed to infantile convulsions or epilepsy, dentition appears to be distinctly a time of peril. Still has seen attacks of *petit mal* which were much aggravated by dentition and ceased when the last tooth was erupted.

(*e*) Rise of temperature. This may occur without the presence of an inflamed condition of the gums, and is probably in most cases purely reflex and analogous to cases in which neurotic children will show a rise of temperature with any excitement, such as a visit to a pantomime ; the rise of temperature with the worry of an erupting tooth is thus part of the induced instability of the nervous system.

(*f*) Attacks of vomiting sometimes occur, with little or no disturbance of the bowels, shortly before the appearance of a tooth.

(*g*) Bronchitis and diarrhœa. Although children are prone to these conditions, there are cases, nevertheless, which seem to be distinctly nervous and traceable to reflex influence from the teeth. That such a condition could exist is quite conceivable, since the bronchial catarrh of the asthmatic child and the lienteric diarrhœa (the nervous diarrhœa of Trousseau) are admittedly dependent in some instances upon nervous influence, often quite remote from the organs affected.

(*h*) Violent screaming attacks, independent of any digestive disorder and not obviously due to any pain, are sometimes associated with dentition. This occurrence is, however, uncommon.

(*i*) Photophobia. This may be so pronounced 'that the child will cover his eyes with his hands or bury his face in the pillow or against his mother's breast, when exposed even to the moderate light of a sunless day.' The photophobia may be so extreme that the child cries or screams violently when exposed to ordinary daylight.

(*j*) Head retraction. Still has seen this symptom causing suspicion of meningitis where teething seemed to be the only cause. This head retraction is often present in cases of increased tension in the middle ear, but he remarks that there has been nothing to suggest ear trouble in the cases he has seen associated with dentition.

(*k*) A rare disorder which is closely related to dentition is *spasmus nutans*, or head-nodding with nystagmus. This disorder is so distinctly coincident with dentition in its onset and cessation, and is so definitely aggravated by the eruption of a fresh tooth, that there would seem to be a causal relationship between the two.

(*l*) Flushing of one cheek, often on the side on which the gum is bulging over a coming tooth, is a curious reflex vasomotor effect of dentition.

Treatment.—The treatment of these conditions lies within the province of the general practitioner, but the dental surgeon may be consulted with a view to lancing the gums. This operation, if considered necessary, should be performed as follows: 'A curved bistoury should be used, and, in order to prevent injury to surrounding parts, a strip of lint should be wrapped round the knife so that only half an inch is exposed. The technique of the operation is quite simple. The child should be placed on a pillow on the lap of the nurse, who should be seated on a chair opposite the operator, and with her back towards the source of light which should come preferably from a north window. The operator should seat himself facing the nurse, with the end of the pillow supporting the child's head in his lap. He thus commands the territory of operation, and, by holding the child's head, can guard against any sudden movement. The hands and the body of the child must be firmly held by the assistant. The lancet is then passed through the overlying tissue until it is felt to come into contact with the enamel surface, and the tissue divided sufficiently to allow the tooth to erupt without resistance' (Kirk). The child's bladder should be emptied before the commencement of the operation.

The best method is to make two semi-lunar incisions which meet at their extremities, and remove the intervening portion of gum with a pair of tenaculum forceps. Objection has been raised to the use of the lancet while the tooth is still considerably below the surface, on the ground that cicatricial tissue offers greater resistance to the progress of the tooth than fibrous tissue; but as cicatricial tissue is of a lower degree of organisation than normal fibrous tissue and therefore more easily disintegrated by pressure, this objection is not tenable.

(2) Local.

Simple Stomatitis.—The stomatitis is usually confined to the neighbourhood of the erupting tooth, and is characterised by intense redness and swelling, the mouth at the same time being hot, the child fractious, restless, and in evident pain. The temperature may reach 104° or 105° F.; but it should be remembered that pyrexia in children readily supervenes upon slight causes.

Ulcerative Stomatitis.—In these cases the gums become hot, swollen, and painful, and these symptoms are especially marked over a certain tooth. Ulceration supervenes, and may extend to the gum around any other tooth already erupted. The ulcers thus formed have a sloughy appearance; the breath is fœtid and hot; the flow of saliva is increased, and the child rejects its food. In addition, there is marked pyrexia and, at times, gastro-intestinal disturbance. Ulcerative stomatitis is due to local infection, and may often be traced to dirty feeding-bottles or neglect of oral hygiene. It is interesting to note that the local disturbances associated with dentition decrease in proportion to the care exercised in dealing with oral hygiene.

Treatment.—Treatment of the local condition consists in paying strict attention to the hygiene of the mouth and the application to the gums of hydrogen peroxide; a brisk purge, such as 1 grain of hydrarg. cum creta., should also be given. In cases where the ulceration shows signs of spreading, the application of an escharotic is advisable. The patient should be isolated.

Cysts of eruption.—Small cysts occasionally form over erupting teeth, but they do not usually interfere with eruption. (See Chapter XXI for details.)

The local disturbances accompanying eruption of the permanent teeth are chiefly associated with the third molars, especially the mandibular. When a mandibular third molar erupts the anterior cusps appear first, and the small portion of gum which lies over the posterior part of the tooth occasionally ulcerates from constant pressure of the antagonising tooth. The ulceration may become extremely painful and the adjacent tissues may become involved. The patient complains of pain in the region of the fauces, but perhaps the most tender point is where the mucous membrane of the gum becomes continuous with that of the cheek. This condition is best relieved by free incisions, care being taken that the knife divides all the tissues overlying the buccal surface of the tooth. Should this treatment not bring relief, it is advisable to remove the process of gum that is covering the tooth; this can be accomplished with the scalpel and forceps. In addition, fomentation of the inside of the mouth should be advised, and hot water at a temperature just bearable or a warm solution of 1 in 80 carbolic acid is, perhaps, as comforting an application as any, the carbolic acid acting as an analgesic. Aspirin gr. x. may also be given, particularly on retiring, as it lessens pain and promotes sleep. Suppuration may supervene, and in that case the offending tooth must be removed, otherwise inability to open the jaws may occur. This may be produced by muscular spasm (trismus), due to reflex irritation ; but it is more than probable that it is generally caused by spread of inflammation to adjacent tissues. The insertion of the temporal and pterygoid muscles and the intervening cellular tissue may, by a process of continuity of inflammation, become affected, and produce this condition. The latter explanation of the occurrence of this inability to open seems to be more in harmony with the clinical aspect of the majority of cases met with, for, in nearly all of them, the patient is able to effect slight movement in the articulation, which would not be possible if it were due to tonic spasm of the masseter muscle. Moreover, the mobility of the articulation increases as the inflammatory symptoms subside. It appears probable, therefore, that in most cases of inability to open, the condition is to be attributed to the inflammatory process, but no doubt instances may occur where it is due to a tonic spasm of the muscles of mastication. The treatment of the above condition is to remove the erupting tooth (see also Ludwig's angina, pages 451 and 455).

Trismus.—By trismus is meant a spasmodic muscular closure of the jaws. Such spasm may be tonic (continuous) or clonic (intermittent). Tonic spasm of the jaws occurs in tetanus, strychnine poisoning, patients going under anæsthetics, hydrophobia, occasionally in cases of fracture of the jaws, and possibly, though rarely, from the irritation of an unerupted mandibular third molar. Clonic spasm of the jaws occurs under such circumstances as the chattering of the teeth from cold or fear.

The word trismus is apt to be loosely used—for instance, it is often used when inability to open the jaws is meant (see p. 991).

The mandibular third molar, when impacted, may give rise to severe odontalgia in the second molar, the pressure from the erupting tooth causing absorption of the posterior surface of the second molar, leading to exposure of its pulp. The absorption in these cases is due to multi-nucleated cells which are developed at the point of contact, and are functionally osteoclasts. In these cases the second molar should be removed. For further reference to misplaced third molars see Chapter IV.

(B) ACCELERATED ERUPTION.

Evidence is accumulating that *endocrine influence* may give rise to changes in the time of eruption of the teeth.

Hyperthyroidism.—It is well known that in thyroid deficiency cases, such as cretins, eruption may be delayed; in the rare cases in which the converse happens and *hyperthyroidism* occurs in children eruption has been found to be accelerated; thus in a case of juvenile hyperthyroidism recorded by H. Middleburgh,[1] a female child aged $4\frac{11}{12}$ was found to have $\begin{array}{cc|cc} 6 & 1 & 1 & 6 \\ \hline & 21 & 12 & \end{array}$ of her permanent dentition erupted.

Adreno-genital syndrome.—Similarly in the rare juvenile cases of the *adreno-genital syndrome* (Richardson and Doll [2]), in which cortical hyperplasia of the adrenals occurs, the teeth in a female child aged $6\frac{1}{12}$ were found by Rushton to be in the state of those of the normal child of ten years, the growth of pubic and axillary hair being that of an adult.

Macrogenitosomia præcox.—Gardiner-Hill and Richardson [3] report the case of a boy aged $8\frac{9}{12}$, one of twins, the subject of *macrogenitosomia præcox*, in whom no lesion could be found; they investigated particularly the possibility of pineal, cerebral, adreno-cortical, testicular and prostatic abnormalities, as lesions in these localities have been known to be associated with this condition. The boy began teething early, but at the age of three all teeth were removed on account of caries; at $8\frac{9}{12}$ his dentition was that of a normal child of ten or eleven years. The authors state, however, that testicular interstitial cell hyperplasia could not be excluded as a possible cause without biopsy, and this had not then been done.

Congenital syphilis is thought occasionally to accelerate the eruption of teeth, though usually it is believed to delay eruption.

Early eruption, usually of individual teeth, may occur as a variation within the range of normal in a child in whom no other abnormality can be found; permanent first molars, for instance, may be found erupted before the age of five years.

The eruption of permanent teeth is often accelerated by early loss of the deciduous teeth, though this, by allowing early forward movement or tilting of teeth as they erupt, may also delay the eruption of later teeth by lessening the space necessary for them so that they are unable to take up their normal position in the arch.

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[1] *Brit. Dent. Journ.*, 1939, lxvi, 294.

[2] *Brit. Med. Journ.*, 1939, i, 501.

[3] *St. Thomas Hosp. Reps.*, 2nd Series, 1939, iv, 35.

Occasionally one or two of the deciduous teeth, usually mandibular incisors, may be erupted at birth ; when this is so the roots are not formed and the teeth are sometimes loose. History records that Louis XIV of France was born with two teeth erupted which had the effect of lacerating his nurse's nipples.

(C) RETARDED ERUPTION.

Rickets.—In children suffering from rickets, the eruption of the teeth is sometimes considerably retarded, and it is not uncommon for the advent of dentition to be delayed until the commencement of the second year. Retarded eruption of the permanent teeth is also met with in those who have suffered from rickets in infancy. The retarded eruption associated with rickets is attributed by some observers to thickening of the tooth sac. The fact that fibrous odontomes are formed in rickety animals supports this view.

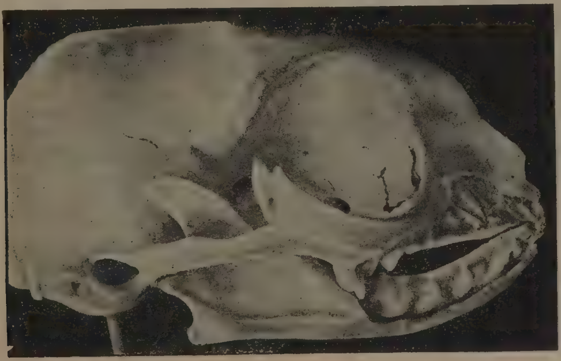

Fig. 1. (From *Trans. Odonto. Soc.*)

The specimen shown in fig. 1 would seem to throw some light on the subject. The animal, a cat which died at the age of three years, had suffered from pronounced rickets. The only teeth present in the mouth at the time of death were an incisor in the maxilla and an incisor in the mandible; the cusps of a premolar and a molar in the maxilla and a molar in the mandible had also appeared.

Considerable difficulty was experienced in removing the outer alveolar plate to expose the teeth. The bone bordering the oral cavity was of a hard, compact nature, and the premolars, instead of resting in crypts, were found to be attached to the bone, the latter being quite ivory-like in character. The maxillary canine rested in a crypt the walls of which were of a hard, compact nature.

Syphilis.—The general effect of syphilis on eruption is to delay the process, but in a few instances syphilis may accelerate eruption. (For further details of the effect of syphilis on dentition see p. 47.)

Idiocy and thyroid deficiency.—The teeth of micro-cephalics and cretins are usually small, and eruption is considerably delayed. In cretins the eruption of both the deciduous and permanent dentitions is often delayed and may also commence irregularly, though in the subsequent growth of the permanent series the trend is towards eventual perfection of the dentition.

It is interesting to note that the administration of thyroid extract, which assists growth in general, also promotes the eruption of the teeth. In cases where no general improvement follows the administration of thyroid, there is no effect on dentition.[1]

Retardation in the Eruption of Individual Teeth.—Occasionally dentition occurs at the normal periods, with the exception of individual teeth which appear only after a considerable interval.

In the maxilla the eruption of the permanent canine may be delayed until middle life ; for example, in two cases these teeth did not appear until the age of thirty-five and forty years respectively, or they may even be present and not erupt at all. Retarded eruption of the canines is more common in the female than in the male. The premolars are, at times, retarded in their eruption. In one patient under observation the maxillary right second premolar did not appear until the age of thirty-five. The third molar is frequently retarded and may not erupt until quite late in life.

In the mandible the eruption of the first and second premolars is frequently retarded, and the appearance of these teeth may be delayed for two or three years. The second premolar is more frequently retarded in its eruption than the first. The mandibular third molar may erupt at any time after puberty. In one case under observation the mandibular right second incisor did not appear until the fourteenth year.

When a tooth appears late in life the process must be looked upon not as an eruption but as an uncovering of an embedded tooth through atrophy of the superincumbent tissues. In these cases infection of the tooth sac may occur, and the condition has been taken for malignant disease.

Apart from the above, minor degrees of retardation of the eruption of certain teeth occur not infrequently as a variation within the range of normal, permanent second molars occasionally not erupting till the age of fourteen.

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[1] J. G. Turner, *Trans. Odonto. Soc.*, xxxiv, 1.

(D) INCOMPLETE ERUPTION.

In the deciduous series it is occasionally observed that the molars have only partly erupted, the process apparently being prematurely arrested. The molars occlude, but the maxillary incisors may overlap the mandibular teeth and impinge on the gum. When the permanent first molars erupt the 'bite' becomes more open and a space is formed between the deciduous molars, as seen in fig. 2. The condition is not infrequently associated with a history of rickets.

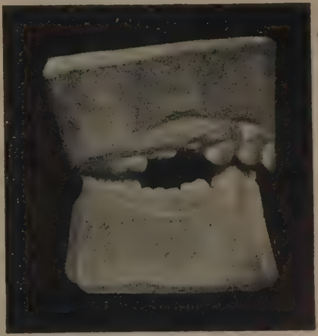

FIG. 2.
(From *Proc. Roy. Soc. Med.*)

This incomplete eruption of the deciduous molars may be limited to the first molars, so that with the eruption of the second molars the first molars will not occlude.

Incomplete eruption of one deciduous molar is rare (fig. 3). In two cases the mandibular second molar was affected and the permanent first molar had tilted forwards. In one case there was a history of injury to the soft tissues from the use of forceps at birth.

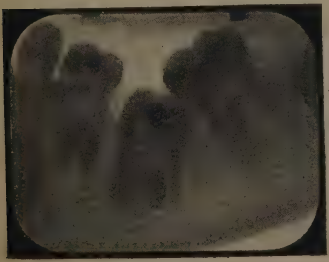

FIG. 3.

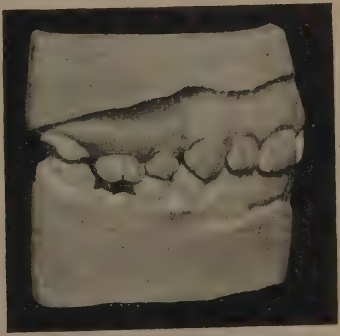

FIG. 4. (From *Proc. Roy. Soc. Med.*)

In the permanent dentition incomplete eruption of the premolars or of the molars may occur, and in such cases there is frequently a history of retarded eruption. In fig. 4 is shown a case where the mandibular right first molar was incompletely erupted.

An instructive case of incomplete eruption of teeth has been recorded by A. T. Pitts.[1] A male, twenty-three years of age, presented the following condition: on the left side of the mandible the first premolar was missing, the second premolar and first and second molars were partly erupted, the crowns of the teeth alone being exposed; on the right side the first molar in the mandible was carious, but sufficient of the crown remained to show that it had only partly erupted, while the maxillary second molar showed a similar condition (see fig. 5).

The radiogram of the mandible showed that the partly erupted teeth were fully formed, but that the body of the bone in this region was insufficiently developed. This would seem to suggest that the eruption of the teeth is closely associated with the growth of the body of the bone, and that when growth is insufficient neither the formation of the root nor the development of the bone around the teeth suffices to bring about the eruption of the teeth.

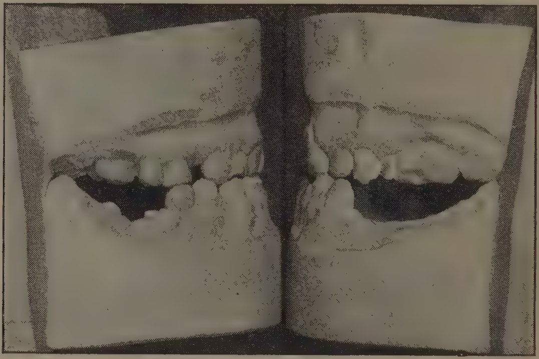

Fig. 5. (From *Proc. Roy. Soc. Med.*)

Teeth which are incompletely erupted are liable to become the seat of chronic sepsis. In the fully erupted tooth the 'gingival trough'[2] is shallow, in the partially erupted tooth it is deep (see diagram, fig. 6).

A deep 'gingival trough' encourages the accumulation of septic material which may cause injury to the periodontal membrane and lead to changes in the hard tissues of the tooth, as illustrated in the case shown in fig. 7.

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), v, 147.

[2] The furrow around the tooth formed by the fold of gum has been named by Hopewell-Smith the 'gingival trough.' This term will be used throughout the following pages.

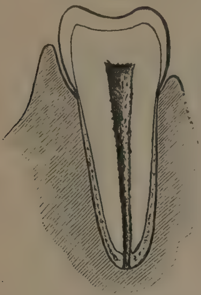

FIG. 6.—The 'gingival trough' in the fully erupted tooth is shown on the right side, that of the partially erupted tooth on the left side.

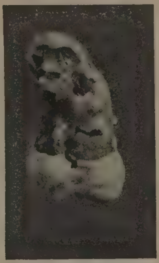

FIG. 7.—Mandibular left second molar showing extensive destruction of the root. (A. Hopewell-Smith.) (From *Proc. Roy. Soc. Med.* (Odonto. Sec.), iii, 9.)

(E) IMPEDED ERUPTION.

This condition may arise in a variety of ways. Premature loss of a deciduous tooth may be the indirect cause of impeded eruption of its successor owing to the tilting together of the adjacent teeth. The obstructing agent may be a supernumerary tooth, or may be a persistent deciduous tooth which has become wedged in consequence of the approximal teeth having inclined towards each other. A pulpless deciduous tooth may also act as an obstruction to its successor, so also may chronic abscesses and cysts formed in connection with such deciduous teeth. Incomplete dichotomies of the deciduous teeth may act as impediments, and a case has been seen in which the mandibular first incisor was prevented from erupting because absorption had taken place only in the root of the deciduous first incisor and not in that of the deciduous second incisor, which was firmly embedded and unabsorbed.

When the deciduous first incisors persist after the eruption of the permanent second incisors the presence of supernumerary teeth may be suspected. In such cases the position and also the condition of the permanent incisors and supernumerary teeth should be ascertained by radiography. If the permanent teeth are found to be normal in shape the supernumerary teeth, together with the deciduous incisors, should be removed, the interval between the second incisors being retained until the permanent first incisors have erupted. Should there be insufficient room in the arch for the permanent first incisors the necessary space should be obtained by extraction or expansion of the arch. On the other hand, if the roots of the permanent first incisors are found to be deformed it is improbable that they will assume a normal position in the arch, and if the supernumerary teeth are of good shape the best course is to allow the supernumerary teeth to erupt and then remove the unerupted incisors.

(F) FAILURE OF ERUPTION.

In the section dealing with retarded eruption reference was made to the fact that teeth may fail to erupt in those who have suffered from rickets. Teeth which have been injured during the period of growth, or have developed in abnormal positions, may remain embedded in the bone.

Cleido-cranial dysostosis.—In this syndrome many of the teeth of the normal dentition may remain unerupted till late in life, and it has been pointed out by Martin Rushton[1] that the condition is often associated with the presence of supernumerary teeth. In one of his cases, a woman aged thirty, the erupted teeth were $\frac{8\quad 4\qquad \mid\ 4\quad 7}{7\ 5d\ 21 \mid\ 1\quad 5}$ and the unerupted $\frac{53\ 21\quad \mid\ 123\quad 58}{8\quad 43\quad \mid\ 234\quad 78}$; there were also one supernumerary tooth in the premaxillary region, two in the mandibular incisor and two in the mandibular right premolar region. Cases of this nature have also been recorded by Lewin Payne and Frölich, and probably Hopson's case recorded in *Proc. Roy. Soc. Med.* xiv. 24 is of this nature. Dentigerous cysts also occurred in Frölich's and one of Rushton's cases. The dysostosis is hereditary and the unerupted teeth present structural defects described on p. 46. Sometimes only a partial syndrome occurs and the dysostosis is limited, as, apparently in two cases, occurring in a mother, aged forty-five, and her son aged sixteen, recorded by Lubner,[2] in which teeth in excess of the normal number occurred and the majority of the permanent teeth remained unerupted ; in the son, bony union had not taken place at the symphisis, and this

[1] *Guy's Hosp. Reports*, 1937, 87, 354.

[2] *Brit. Dent. Journ.*, 1937, lxiii, 2, 205.

occurred in two of Rushton's cases. A daughter aged fifteen was normal; both mother and son were of normal intelligence but of small physical development. The son when examined showed erupted and present $\begin{array}{r|l} 6\quad edc & de\quad 6 \\ \hline 65\ \ 3\ \ 21 & 12\quad 56 \end{array}$, but radiological examination

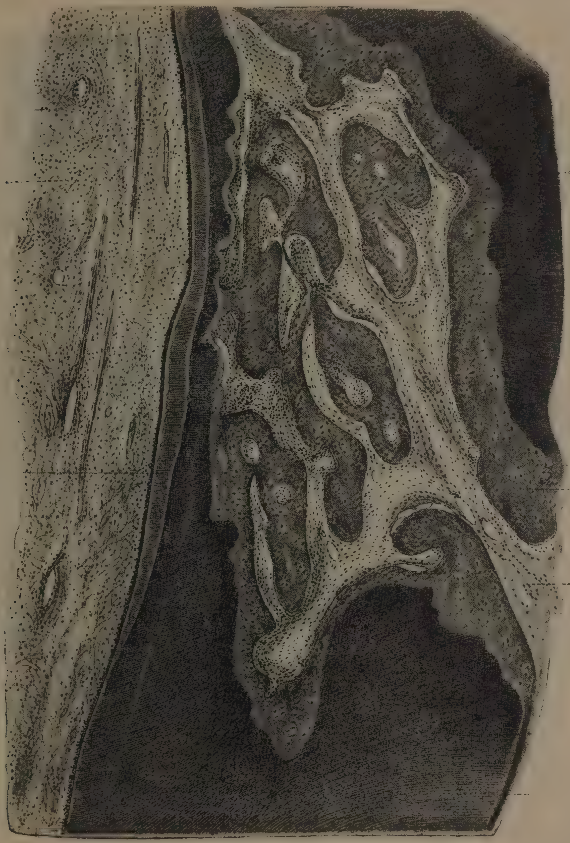

FIG. 8. (From Ash's *Quarterly Circular.*)

showed $\begin{array}{r|l} 87\quad 5443221 & 1223445\quad 78 \\ \hline 7\quad 44\quad & 344\quad 7 \end{array}$ present but unerupted. His mother appeared edentulous, and had been wearing full dentures for many years, but radiological examination showed $\begin{array}{r|l} 5\quad 3221 & 122345\quad 7 \\ \hline 8\ \ 544321 & 1234445\quad 8 \end{array}$ present but unerupted.

Pathological changes of structure often occur in teeth which have failed to erupt.—The changes consist in an absorption of the dentine and occasionally of the enamel; sometimes there is a replacement of the lost substance by tissue of an osseous character, producing a condition of anchylosis (figs. 11 and 12). This is almost always accompanied by an excess production of cementum, produced possibly by inflammation brought about by resistance to the normal stimulus which should cause the tooth to erupt.

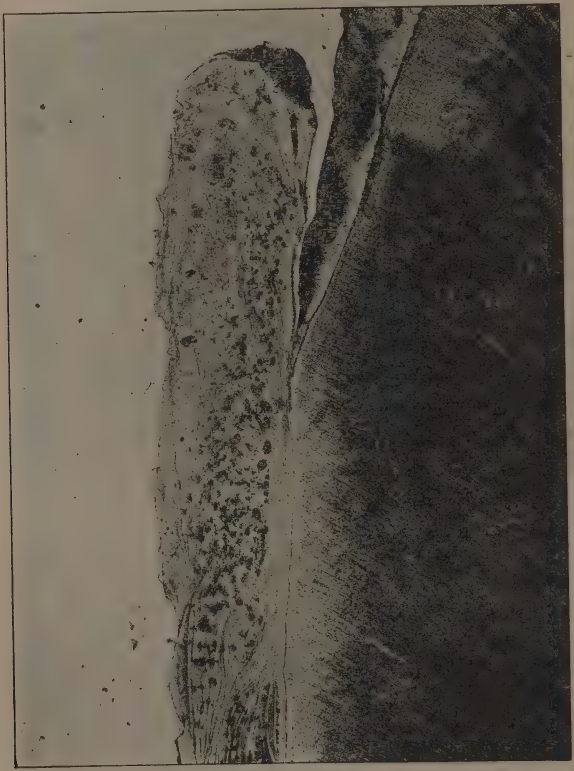

Fig. 9.—Longitudinal section. × 50. (From *Proc. Roy. Soc. Med.*)

A case illustrating these changes is described by Williger.[1] An unerupted maxillary canine was removed, and, on section, spots were visible as if the dentine were perforated. On microscopical examination the dentine was seen to have been absorbed and in great measure replaced by osseous tissue (fig. 8). The absorption was due to a growth of tissue from the periodontal membrane and not from the pulp. The presence of secondary dentine on the walls of the pulp canal showed that the pulp had reacted in the same manner as in other injuries of the dentine.

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[1] Translated in Ash's *Quarterly Circular*, January 1909, from the *Correspondenz-Blatt für Zahnheilkunde.*

An interesting case is recorded by A. W. Baker[1] of an unerupted mandibular third molar which showed definite changes.

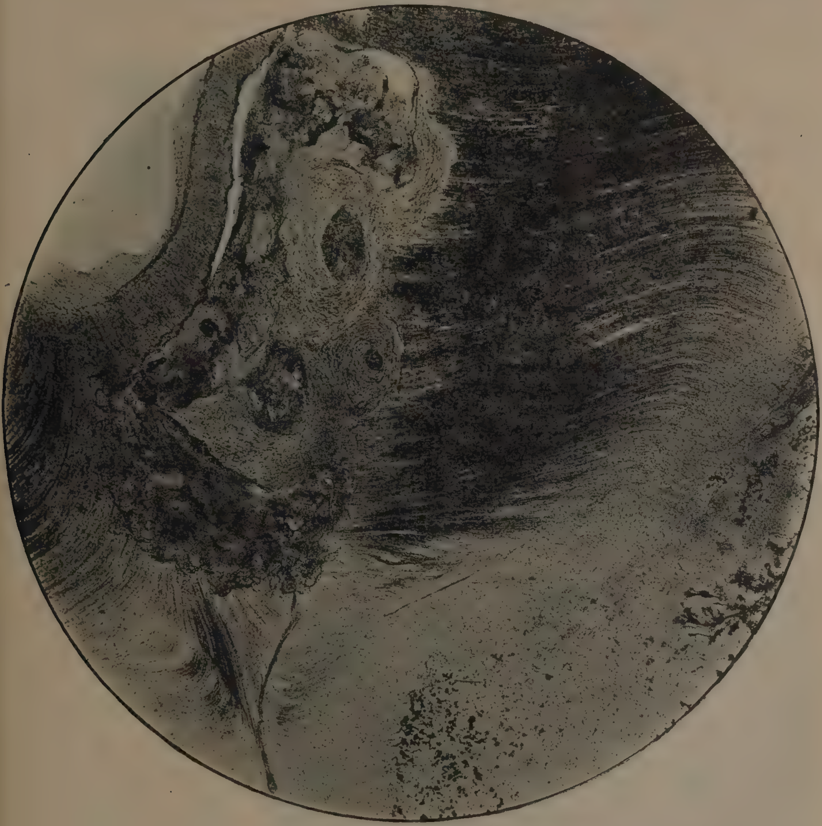

FIG. 10.—Transverse section. × 60. (From *Proc. Roy. Soc. Med.*)

A sinus led to the tooth, and it is probable that there had been infection of the tooth crypt. The tooth showed considerable hyperplasia of the cementum, a thick layer of this tissue having been deposited external to the enamel and the original cementum, as shown in fig. 9. At one part of the root the original tissue had been removed and, in parts, replaced by compact bone (fig. 10).

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), i, 130.

In Williger's case, quoted above, there is no mention of the presence of sepsis, but in the cases which have come under one's personal observation infection has usually been present, and the changes which occur in misplaced teeth are probably often the result of long-continued infection of mild intensity.

In the case illustrated in fig. 11 the enamel and dentine of the crown of the unerupted canine had been partly absorbed and the lost tissues partly replaced by bone, to which they are attached. Fig. 12 shows the absorption of enamel and dentine radiologically in an unerupted canine.

FIG. 11.—Unerupted maxillary canine from an adult; absorption of enamel has been followed by bony anchylosis. (London Hospital Dental Museum.) (E. S.)

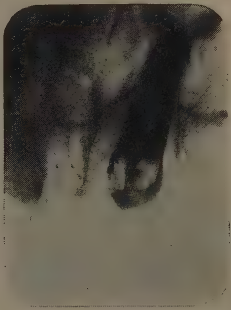


FIG. 12.—Unerupted maxillary right canine, male, aged 57. On extraction it showed absorption of enamel and dentine. (W. Peebles.)

CHAPTER II

Variations of the Teeth.

Variations in Size—Variations in Number—Variations in Shape—Variations in the course of the Blood-vessels—Variations in Colour.

(A) VARIATIONS IN SIZE.

There is considerable variation in the general proportions of series of teeth. See fig. 13.

As a rule the teeth in a normal and complete dentition bear a definite proportional relation to one another in size. Occasionally, however, this relation is disturbed, and one or more teeth will be found to be disproportionately large or small in comparison with the remainder of the series.

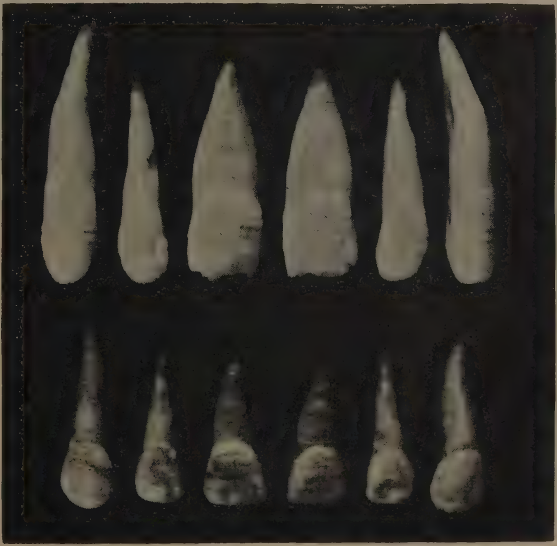


Fig. 13.

In other cases the dentition as a whole is composed of teeth larger or smaller than normal; a dentition composed of teeth larger than normal is commoner than the opposite condition, and occurs more frequently in males. A condition of large teeth crowded in the arch is more common than that of small teeth spaced. The size of the dentition as a whole usually bears some relation to the size of the individual, but this is not always so.

The teeth also vary in size in different races, which have accordingly been classified into Microdont, Mesodont, and Megadont races. The measurements for such classification are:

$$\frac{\text{Length of teeth} \times 100}{\text{Basion to Nasion}}, \text{ and the result is termed}$$

the Dental Index and classifies races as follows :—

Microdont 42 Europeans,
Mesodont 43 Chinese, Malays, Negroes,
Megadont 44 and over, Australian aboriginals.

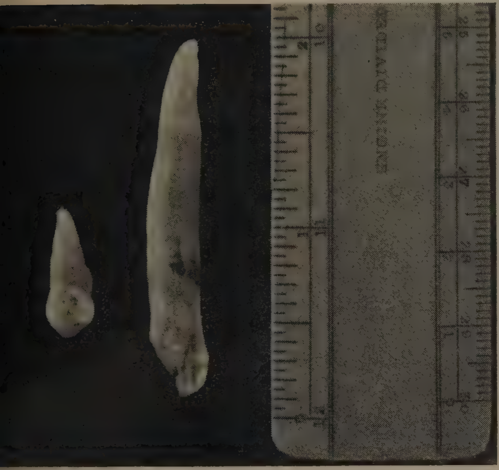
This really compares the size of the skull with the masticating area and is influenced by the degree of prognathism present, so that there is some correlation between the size of the teeth (and hence arch) and the degree of prognathism present.

The size of the deciduous dentition as a whole is usually closely correlated with that of the permanent dentition.

(1) Deciduous Dentition.—The maxillary canine or mandibular second molar may be proportionately larger than the other teeth. Such teeth have no particular interest unless they persist, in which case it is sometimes difficult to distinguish them from the permanent teeth. If, however, care is taken in the examination of the mouth, and the history of former extractions is ascertained, no error is likely to arise. Variations in size are not so common in the deciduous as in the permanent dentition. A large second deciduous molar from one mouth may be larger than a small permanent first molar from another mouth.

(2) Permanent Dentition.—In the permanent series, the tooth which is most frequently developed to an *abnormally large* size is the maxillary first incisor. Such teeth should not be mistaken for a first incisor geminated with a second incisor or having a dichotomous outgrowth. The mandibular second premolar and the mandibular second molar are, occasionally, abnormally large. Not uncommonly the crown of the mandibular third molar is disproportionately large, both to the rest of the dentition and the extent of its own roots. *Diminution in size* occurs mostly with maxillary second incisors. These teeth may also be modified in their shape, and, in extreme cases, are represented by simple cones. The maxillary and mandibular third molars, like the second incisor, are occasionally dwarfed and modified in shape.

Under this heading it is convenient to include those rare cases of hypertrophy of teeth seen in connection with hypertrophy of the jaws. Fig. 14 represents the models of a boy with an enlargement of the right side of the face, including the right half of the tongue. The frontal bone was involved, a distinct ridge running vertically down the centre of the forehead. In the affected maxilla the premolars had not erupted and the canine and first molar were enlarged; in the mandible the canines, premolars, and molars were larger than the teeth on the left side. (See also p. 102.)



IG. 13A.—Two maxillary left permanent canines, both from females aged about 30. The small one was unerupted and symmetrical with the unerupted right canine. The large one was from a patient having undue hairiness of left side of body, trunk, limbs and face; there is no history as to dental symmetry. (E. S.)

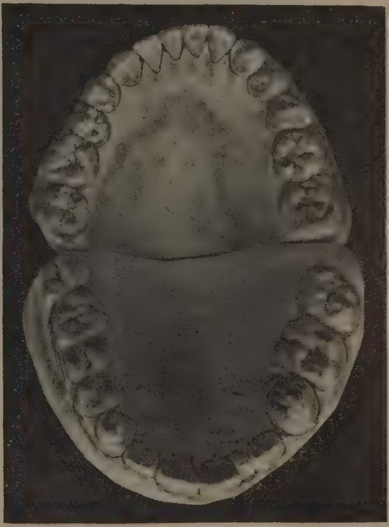


Fig. 14.—Models showing unilateral hypertrophy of teeth.

(B) VARIATIONS IN NUMBER.

The number of teeth in a complete dentition may be greater or less than the normal number. Teeth in excess of the normal number have been dealt with in Chapter XXII.

The term *Anodontia* is used to denote complete absence of teeth, but it is far more common to find one or a few teeth absent only, that is, a condition of *Local anodontia*.

Local Anodontia.

Deciduous Dentition.—Deficiency is not so common as an excess in number. The missing tooth is usually the maxillary second incisor, but all incisors may be absent. A model of a maxilla with both second incisors missing is shown in fig. 15. The maxillary second molars are at times absent, and one has also found the maxillary first molars, and mandibular second molars, absent in different cases. In the case where the maxillary first molars were absent two premolars subsequently erupted on each side, all of which were rather smaller than normal; but sometimes there is absence also of the successional tooth. The condition may be hereditary. The absence of a tooth cannot be definitely determined except by means of radiography.

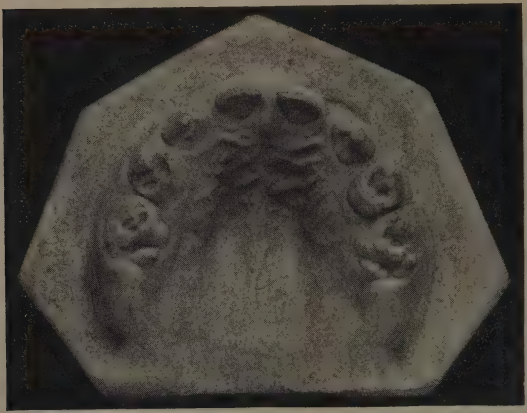


Fig. 15.—Model showing absence of both maxillary deciduous second incisors. (E. S.)

A few cases of the entire absence of the deciduous teeth or their absence in one jaw only have been recorded. The absence of mandibular incisors has been recorded, the absence being repeated in the permanent dentition; but in a recent (1941) case seen by one of us, in which a boy aged four had congenital absence of mandibular deciduous first incisors, X-rays showed all the permanent incisors to be present; the boy had no dental caries.

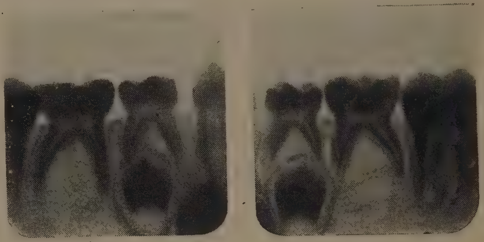


Fig. 15a.—Female aged $6\frac{4}{12}$ years. Radiograms showing congenital absence of $\overline{5\,|\,5}$. (E. S.)

Permanent Dentition.—Any number of teeth may be missing—that is to say, the deficiency may be limited to a single tooth or may extend to the whole series, but the commonest condition is that in which the deficiency is limited to one or two teeth. The condition is sometimes hereditary.

The maxillary second incisors are most frequently missing; after these, the mandibular second premolars (fig. 15A) and third molars, the maxillary third molars, and the second premolars. When the third molars appear to be missing, care must be taken to ascertain that the teeth supposed to be the first and second molars are not in reality the second and third molars, the first having been lost at an early age.

The absence of a maxillary second incisor on one side is often associated with mal-development of the corresponding tooth on the other side. Absence of the second incisors is met with in skulls of semi-civilised and barbaric races, and Wright[1] records such a case in the skull of a neolithic man. R. Clement Lucas[2] states that hare-lip and cleft palate in the child are often associated with the absence of the second incisors or with an ill-developed incisor in one of the parents. A paper on heredity as it concerns the dwarfing or absence of the maxillary second incisors, by R. Bradlaw,[3] illustrates many family trees showing this abnormality; in one family it was traceable through four generations and in five others through three generations.

With regard to the absence of a premolar, the possibility must be considered of the tooth germ having been destroyed by sepsis or removed during the extraction of the deciduous molar. (See figs. 892A, i and ii.)

The absence of the third molars would appear to be more common in highly civilised communities than in primitive races. Absence of one or both mandibular first incisors is a rare but well recognised condition, and is occasionally associated with absence of the maxillary second incisors. In a case under the care of H. Stobie, the maxillary second and mandibular first incisors were absent in addition to the second premolars.

Absence of a permanent canine from the series is very rare. A case was recorded[4] in which the maxillary left tooth was absent, the deciduous tooth being present. The absence of the tooth was determined by radiography.

Persistence of the deciduous canine is met with in a large number of patients, mostly females. In these cases the permanent successor is usually present, but is situated in an erratic position.

Occasionally, but rarely, absence of a tooth on one side is associated with duplication of the same tooth on the opposite side: such a case is seen in fig. 16. T. Widdowson[1] records another case in which the mandibular left permanent canine was duplicated and the canine absent on the right. Comparable cases also occur where duplication occurs on one side and a malformed tooth on the other: exhibit B.41 in the Gordon Museum (Guy's Hospital Dental School) illustrates this in the maxillary permanent incisor region.

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), ii, 3.

[2] *British Journal of Children's Diseases,* November 19, 1904.

[3] *Dental Record,* 1934, liv, 113.

[4] *Dental Cosmos,* May 1922, 575.

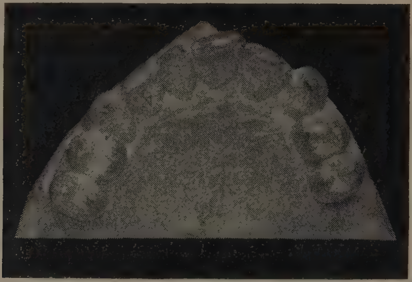

FIG. 16.—Concomitant variation of maxillary permanent second incisor It is absent on the left and reduplicated on the right. (L. Newbald).

The whole group of incisors, maxillary and mandibular, may be absent, occasionally the premolars, and very rarely the molars. In cases where a **large number of permanent teeth** are absent, the first molars are usually present on either side in maxilla and mandible and one or two badly shaped teeth in the incisor region (fig. 17). An instance of marked deficiency of permanent teeth in twins aged sixteen years was observed by T. Clarence. Their dentitions were :—

$$\frac{\mathrm{76\ \ 4c\ 1}}{\mathrm{6\ \ dcba}} \Bigg| \frac{\mathrm{1\ c45\ 7}}{\mathrm{abc\ e}} \quad \text{and} \quad \frac{\mathrm{76\ 4c\ 1}}{\mathrm{dcba}} \Bigg| \frac{\mathrm{1\ c4\ 67}}{\mathrm{abc}}$$

A very interesting case is recorded by Jarre.[2] There were no deciduous teeth until the age of fifteen months, when a tooth, conical in form, appeared in the maxilla on the left side of the median line. Four months later a similar tooth appeared on the right side. At the age of twenty-three months a tooth erupted in the situation of the maxillary second incisor. About the third year two more teeth of the size and form of deciduous molars appeared in the maxilla on the right and left sides. No teeth appeared in the mandible. At the age of seven years the three teeth in the incisor region were replaced by three others, also conical in form. The deciduous molars were persistent at the age of twelve, when the condition of the jaws was as follows: In the maxilla five teeth, three being permanent, two deciduous; in the mandible no teeth. The hair on this patient's head was scanty, while the rest of the body was entirely hairless. The nails presented peculiarities. They were covered with white opaque spots and streaks which occupied the whole thickness of the tissue, this condition having been present since birth. The lenses were normal. The skin was loose and wrinkled. The

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[1] *Brit. Dent. Journ.*, October 1, 1929.

[2] *Dental Cosmos*, June 1892, 468.

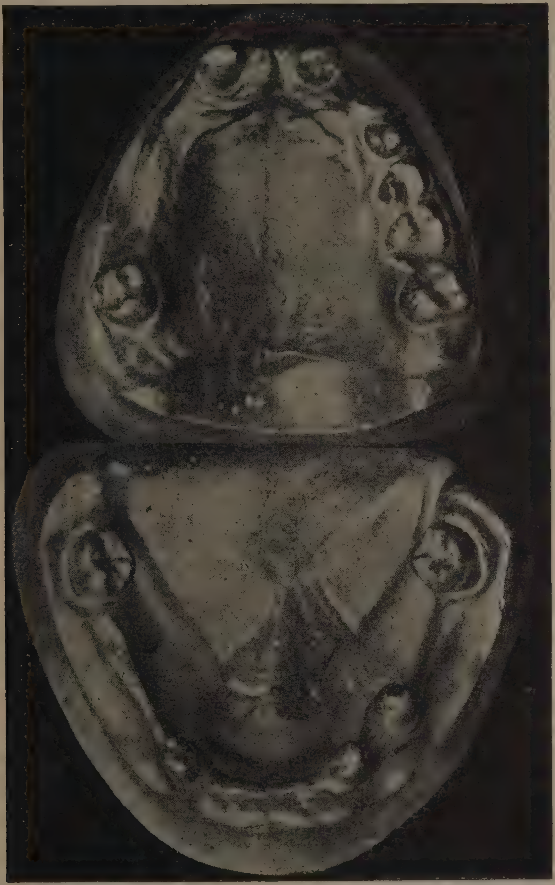

FIG. 17.—Models showing deficiency in the number of permanent teeth. Male, aged nineteen. (E. S.) (From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

coincidence of abnormalities in teeth, hair, skin, and nails is very interesting, as they are all epithelial in origin.

Where there is a marked deficiency in the number of teeth, both sides of the jaws are usually affected, although not necessarily to the same extent. For example, a girl aged nine years had no teeth on the left side of the mandible and only the deciduous canine, deciduous second molar, and permanent first molar on the left side of the maxilla, while on the right side only the first incisors in both maxilla and mandible were missing. The left side of the girl's scalp was almost totally devoid of hair.[1]

ANODONTIA (COMPLETE ABSENCE OF TEETH).

A case of **entire absence of teeth** accompanied by marked deficiency of hair is recorded by Guildford.[2] The patient, a man aged forty-eight, was nearly bald, there being only a slight covering of down. Hair was present in the pubic and axillary regions, but the surface of the body was entirely free from hair. The sudoriferous glands were absent or suppressed, for he had never perspired, and he had no sense of smell or taste. Of his six children, two showed signs of inherited abnormality in having only about half the normal number of teeth.

A case of total absence of teeth in a boy aged sixteen is recorded by W. Rushton.[3] The only other abnormality associated with the condition was in the hair, which was of a soft character resembling wool.

Total absence of teeth in two brothers has been recorded by J. H. Gibbs.[4] There was definite abnormality of the skin, nails, hair, and sudoriferous glands, and a history of defective tooth formation in other members of the family, the defect being transmitted through the female parent.

Battersby [5] records another case in a boy of nine, having fine light-coloured hair, scanty eyebrows and perspiring only at his hands and feet ; his family tree shows that in five generations, ten of his relatives showed local anodontia involving many teeth ; of these his mother, grandfather and great-great-grandfather appear to have had complete anodontia of the deciduous dentition as well. Battersby also refers to other cases of complete anodontia recorded by Nager, and Herbst and Apffelstaedt.

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[1] P. Ledger Etheridge (*Brit. Dent. Journ.*, January 15, 1913).

[2] *Transactions World's Columbian Dental Congress*, 257.

[3] *Trans. Odonto. Soc.*, xxxvi, 17.

[4] *Dental Record*, November 1, 1915, 669.

[5] *The Dental Magazine*, 1936, 53, 427.

In a case recorded by W. Schoenlank [1] the absence of teeth was associated with epithelium showing fœtal conditions in the shortness of papillæ which passed into the underlying connective tissue.

Cases of **absence of teeth associated with abundance of hair** have been recorded. In the case of Andrian Jeftichfen,[2] the whole of the face, nose, forehead, cheeks, and ears was covered with long brown hair, which also extended partly down the back. His son, Fedor, at the age of three years showed the same tendency to hairiness. The father had no teeth up to seventeen years of age, and eventually only erupted four teeth in the mandible and one in the maxilla. The son, at the age of three, possessed only four incisors in the maxilla. The Burmese hairy family, consisting of grandfather, mother, and son, are said to have been deficient in the number of their teeth, but there is no reliable record.

From the foregoing cases it is evident that there is a correlation between the hair and the teeth. In one class there is deficiency of both structures; in the other, deficiency of teeth is associated with redundance of hair.

A case of correlated variation involving skin, hair, and teeth is recorded by Darwin in a Hindu family. Ten men of four generations were affected: the females of this family were unaffected, but they transmitted the tendency to their sons; the condition, therefore, was here a sex-linked recessive characteristic, and as such has also been found among Europeans. Occasionally both sexes in a family may be affected, and the condition is then inherited as a dominant.

Absence of teeth should always be confirmed radiologically, otherwise some cases may prove to be of the nature of cleidocranial dysostosis or, if associated with excessive hairiness, of general hyperplasia of the gums (*cf.* ChapterXVI).

When deciduous teeth are retained owing to the congenital absence of their permanent successors, it might reasonably be supposed that they would remain functional during the remainder of that patient's life. This does occasionally occur. One has known of a functional deciduous tooth at the age of fifty-six years, but usually the roots become absorbed, and they are lost before middle age is reached.

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[1] *Deutsche Monatsschrift für Zahnheilkunde*, January 1921.

[2] *Brit. Journ. Dent. Sci.*, xvi, 527.

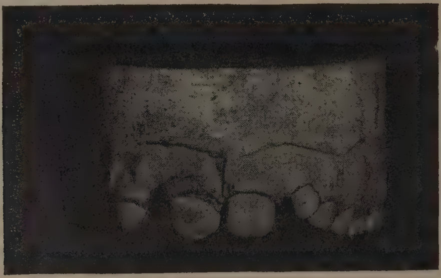

FIG. 18.—Model showing absence of the maxillary right second incisor.

There is no satisfactory explanation of the absence of any or all of the permanent teeth, but the fact that certain teeth are more constantly absent than others lends support to the view that in some cases it may be an evolutionary change. Against this is the fact that an excess in number is slightly commoner than absence. Both may be evolutionary changes, illustrations of the universal tendency to vary, but as neither variation is of advantage or disadvantage under present conditions, both will probably continue to occur either sporadically or as inherited variations.

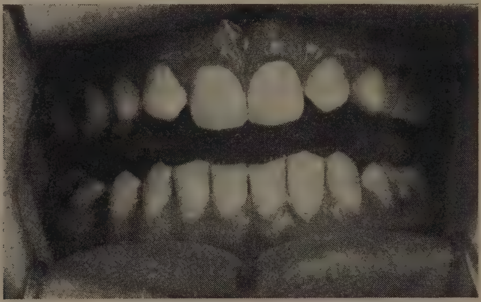

FIG. 19.—Female, age 14. Congenital absence of both maxillary permanent second incisors; exceptionally small permanent canines are adjacent to the first incisors, and the deciduous canines are in line distal to the permanent canines. In this case this arrangement happened spontaneously. (E. S.)

Treatment.—Absence of the maxillary second incisors should be suspected if these teeth have not erupted at the age of ten years. The treatment will depend upon the conditions present.

In the majority of cases the deciduous second incisors and canines should be extracted in order to allow the permanent canines to erupt towards the first incisors.

If only one incisor is absent and the fellow tooth is diminutive the latter may be removed in order to obtain a symmetrical arrangement of the teeth, and their absence masked by stoning away the tips of the canines should they be too noticeably prominent. In the case fig. 18 the right tooth was absent, with a fair interval between the first incisor and the deciduous canine. The radiogram showed that the permanent canine was erupting towards the incisor. Under such conditions, if there is sufficient space for the permanent canine to erupt without causing absorption of the root of the deciduous canine the latter may be allowed to remain (fig. 19); but should the deciduous canine show signs of absorption, its early removal is called for, in order to allow the premolars to move forward.

(C) VARIATIONS IN SHAPE.

The variations here tabulated of the deciduous and permanent teeth, but not those involving many teeth, are well recognised; most of them occur symmetrically, though not invariably so; they interfere in no way with function or occlusion, and so may be taken as coming within the range of normal variation.

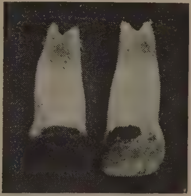

FIG. 20.—Maxillary deciduous first incisors, showing bifurcation of the roots, which are bent forward. (From *Proc. Roy. Soc. Med.*)

(1) Deciduous Dentition.

Cases of pronounced cusps arising from the cervico-marginal ridge of the maxillary incisors and canines are met with. An additional cusp is frequently present at the antero-internal aspect of the maxillary second molar. When this is so, one almost invariably finds an extra cusp in the corresponding position on the maxillary permanent first molar (Carabelli tubercle)[1]; an extra cusp is frequently found on the antero-external aspect of the first molar, both in the maxilla and mandible.

An interesting variation of the maxillary first incisors has been observed and described by A. T. Pitts.[2] The roots, as shown in fig. 20, are bent forward on themselves and are grooved longitudinally, the groove being more marked on the labial surface, and the extremities are bifurcated.

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), xv, 72. (E. S.)

[2] *Ibid.*, v, 41.

The maxillary canines may be peg-shaped and the mandibular teeth may be deeply grooved on the labial surfaces. Occasionally the root of the mandibular canine is bifid. The maxillary second molar may have four roots and the mandibular three. When this latter occurs the extra root is almost invariably in the same position as when this occurs on the mandibular permanent first molar, and on the postero-internal aspect. The roots of the mandibular first molar may be fused together.

(2) Permanent Dentition.

(*a*) Maxillary Teeth.

(i) *First Incisor.*—The following are some of the variations in shape met with :—

(*a*) A considerable flattening from before backwards (fig. 21).

(*b*) The crown altered so as to resemble a simple cone (fig. 22).

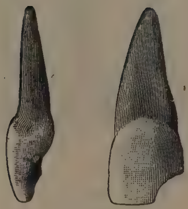

Fig. 21.

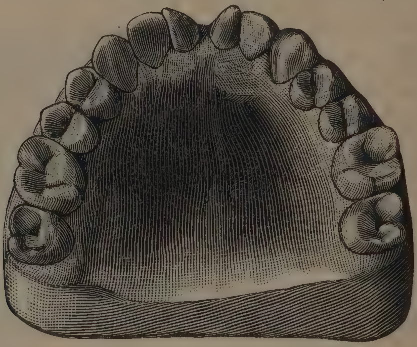

Fig. 22.

(*c*) Disproportion between the width of the crown and the root (fig. 23, *a*).

(*d*) Disproportion between the length of the crown and the root (fig. 23, *b*).

(*e*) Groove running from the posterior cervico-marginal ridge on to the root (fig. 23, *c*).

(*f*) Marked development of the posterior cervico-marginal ridge (fig. 23, *d*).

(*g*) Development of a cusp from the posterior cervico-marginal ridge, the cusp diminishing in size towards the cutting edge (fig. 23, *e*). The formation of the cusp may be associated with a bend in the labial surface of the tooth (fig. 23, *f*).

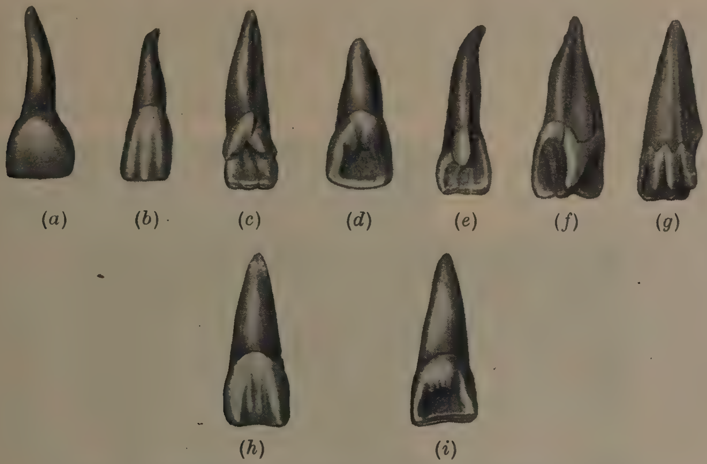

Fig. 23.—Maxillary first incisors showing variations in shape.

(*h*) Development of three cusps from the cervico-marginal ridge associated with marked bending of the labial surface (fig. 23, *g*).

(*i*) A thickening of the mesial and labial margins of the anterior and posterior surfaces (figs. 23, *h*, and 23, *i*).

(*j*) An extra root (fig. 24).

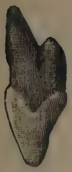

Fig. 24.

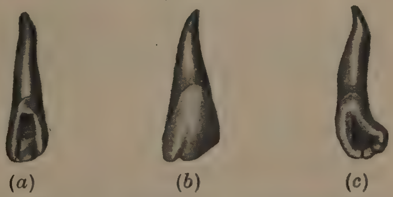

Fig. 25.—Maxillary second incisors showing variations in shape.

(ii) *Second Incisor.*

(*a*) Variation in shape of the crown, the mesial and distal angles being so rounded as to cause the crown of this tooth to resemble a canine. Under these circumstances the roots also may be stouter and more caniniform than normal.

(*b*) The crown so altered as to appear as a simple cone.

(*c*) Pronounced lingual fossa due to development of the posterior cervico-marginal ridge (fig. 25, *a*).

(*d*) A groove running down the medial aspect of the root.

(*e*) Development of a cusp from the posterior cervico-marginal ridge, accompanied in some instances by a groove running down the root.

(*f*) A notch on the cutting edge towards the medial angle (fig. 25, *b*).

(*g*) Depression in the medial aspect near the neck (fig. 25, *c*).

(*h*) An extra root.

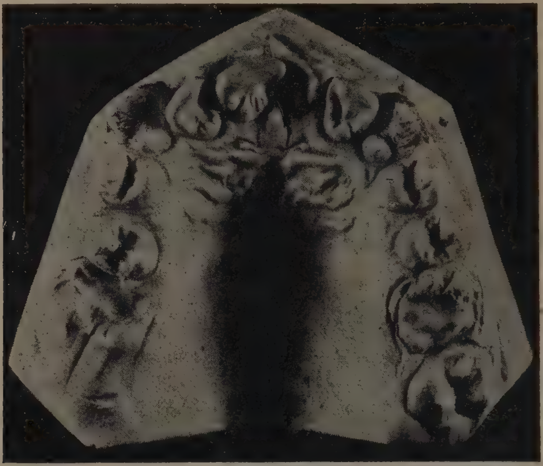

Fig. 26.—Model of maxillary teeth with extra cusps on first and second incisors and particularly large extra cusps on canines. Male. (E. S.) (From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

(iii) *Canine.*

(*a*) The root may be grooved, and bifurcation of the apex may take place. (The latter is very rare.)

(*b*) Development of a cusp from the posterior cervico-marginal ridge. This variation may be so marked as to cause the tooth to resemble a premolar (fig. 26).

(*c*) The crown may be divided into two portions by a vertical cleft, so making it bifid (fig. 27), this is an incomplete equal dichotomy.

(*d*) A spike-like projection from the labial surface (fig. 28).

(iv) *Premolars.*—**The first premolar** is very irregular in regard to the form and number of its roots. Externally the root nearly always shows signs of bifurcation, and in some cases possesses two or three well-defined roots. When there are three roots they are arranged on the same plan as in the maxillary molar, and are usually symmetrical. It is thought this may be atavistic as the maxillary premolars of anthropoids normally have three roots similarly arranged.

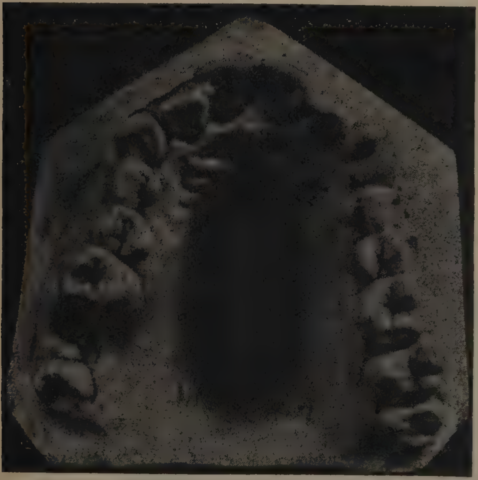

FIG. 27.—Maxillary left permanent canine with bifid crown. Female. (E. S.) (From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

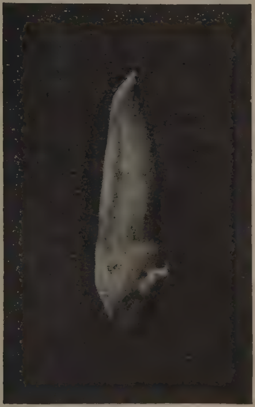

FIG. 28.

(Hunter collection, Royal College of Surgeons, Odonto. series H. 105.)

Three-rooted maxillary first premolars may be associated with a marked tendency to two roots on the corresponding mandibular teeth.

The second premolar is more constant in form than the first premolar. Two or three roots may be present. An extra cusp occasionally appears on the premolars (fig. 29).

(v) *Molars.*—**The first molar** may have a fourth root. The fourth root is always situated between the anterior buccal and the palatine roots. A fifth root is occasionally seen. The roots may be conjoined, a union of the posterior buccal and palatine being most common. At times all three roots may be united (fig. 30). H. P. Pickerill[1] has recorded an interesting variation of the maxillary first molars (fig. 31). The crowns were large but normal in shape; the apices, instead of being conical,

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), ii, 150.

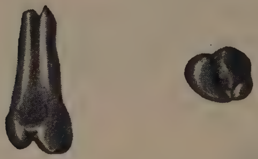

Fig. 29.—Maxillary premolar with an additional cusp.

Fig. 30.—Maxillary permanent first molar with all three roots joined. Female, aged thirteen. (London Hospital Dental Museum.) (E. S.)

presented a large, uneven quadrilateral surface, the naked-eye appearance suggesting that four roots had spread out and had become united, the centre being filled in with cement. A section (fig. 32) showed one large central quadrilateral pulp cavity

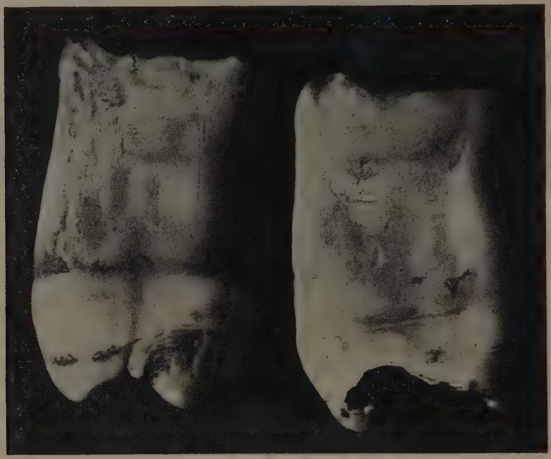

Fig. 31. × 2½.
(From *Proc. Roy. Soc. Med.*)

surrounded by a circular and parallel wall of dentine. Such a condition might be regarded as a form of dilated odontome (see Chapter XXII).

The buccal roots of the maxillary molars are often curved, and may approximate at their apices so as to embrace the septum. An extra cusp is often seen on the mesio-lingual aspect; it is present to a marked extent in about 25 per cent. British, and it, or a trace of it (as a dimple representing the fissure at the junction of this cusp with the crown), may be found in some 65 per cent. British.[1] It is often known as the Carabelli tubercle. It has been thought to be one of the stigmata of congenital syphilis, but it is not so; it occurs also among the Anthropoidea.

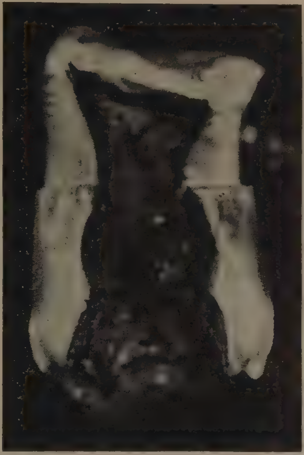

FIG. 32. × 2½.

(From *Proc. Roy. Soc. Med.*)

The second molar may present abnormalities similar to the first. A peculiar flattening of the tooth, as shown in fig. 36, is met with; frequently there is absence of the postero-internal cusp (hypocone), giving the crown a triangular shape. Though an extra antero-internal cusp corresponding to the Carabelli tubercle may occur on this tooth, it is decidedly uncommon.

The third molar assumes a great variety of shapes. The roots may be all joined together, and in one specimen in the Museum of the Royal College of Surgeons the apex is cup-shaped with numerous holes for the vessels of the pulp to enter. The roots may be increased in number, and flexions are often present.

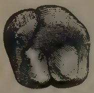

FIG. 33.—Occlusal aspect of normal maxillary left first molar.

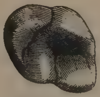

FIG. 34.—Occlusal aspect of oblique-rooted maxillary left first molar.

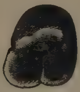

FIG. 35.—Occlusal aspect of normal maxillary right second molar.

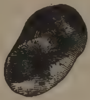

FIG. 36.—Occlusal aspect of oblique-rooted maxillary right second molar.

The normal position of the roots of the molars may be altered by the posterior buccal root being displaced inwards and forwards. This variation is termed 'oblique rooted,' and attention was first drawn to it by Booth Pearsall[1] (see figs. 33 to 36).

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), xv, 72. (E. S.)

(*b*) Mandibular Teeth.

(i) *Incisors.*

(*a*) The crown may be altered so as to resemble a single cone.

(*b*) A notch may be present on the cutting edge (fig. 37, *a*), this apparently being caused by a partial dichotomy of the enamel organ.

(*c*) A thickening may occur on the labial aspect in the median line (fig. 37, *b*).

(*d*) An extra root may arise from the lingual surface.

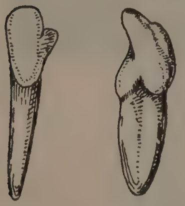
(*a*) (*b*)

Fig. 37.

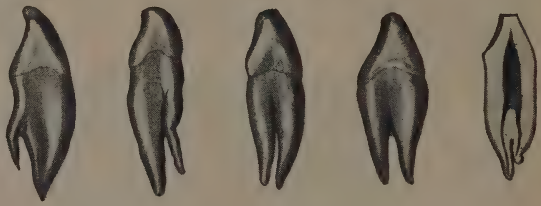
Fig. 38.—Two-rooted mandibular canines.

(ii) *Canine.*—This tooth may have an extra root. Various specimens of this abnormality are shown in fig. 38. G. Harborow[2] has shown that this condition was asymmetrical in seventeen out of twenty cases.

(iii) *Premolars.*—**The first premolar** may have two roots. 'When a lower premolar tends to have two roots they are peculiarly formed, the flattened apex being bent round so as to form an approach to a second (anterior) root, and this may go on to complete division' (fig. 39). It is thought that this may be atavistic as the mandibular premolars of anthropoids normally have two roots also arranged anteriorly and posteriorly. The inner cusp of this tooth is at times feebly developed and the tooth then simulates a canine.

The second premolar is more constant than the first. A curious abnormality of this tooth, which is fairly common, is shown in

[1] *Brit. Dent. Journ.*, 1889, x, 38.

[2] *Ibid.*, March 1934, 244.

fig. 40. The crown has, not uncommonly, as many as five cusps; rarely this tooth is two-rooted, and when this is so one has, on two occasions, found them to be placed lingually and buccally.

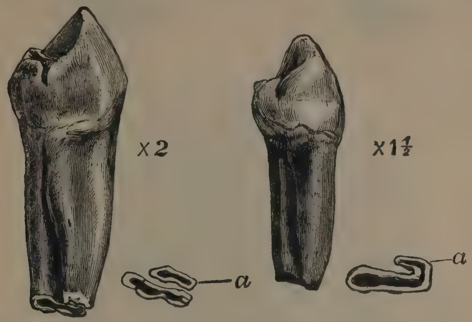

FIG. 39.—Two mandibular premolars, the roots of which show two stages towards complete division. *a*, Anterior root. (From Tomes's 'Dental Anatomy.')

(iv) *Molars.*—**The first molar** may have an extra root. When this is so it is almost always present on the postero-lingual aspect. An extra root on the buccal side of the tooth is rare (fig. 41). Four roots are met with, two being anterior and two posterior.

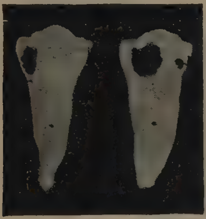

FIG. 40.—Mandibular second premolars from different mouths with crowns much enlarged in antero-posterior diameter and triconodont appearance from buccal aspect. (E. S.) (From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

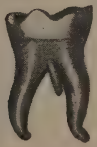

FIG. 41.—Mandibular first molar with an extra root on the buccal aspect.

Occasionally the roots are found to be joined together, and the normal backward curve much exaggerated. Cases of five and six roots, which have been recorded, are possibly examples of gemination. Extra cusps are met with on both buccal and lingual surfaces.

The second molar presents abnormalities similar to those

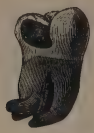

FIG. 42.

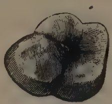

FIG. 43.

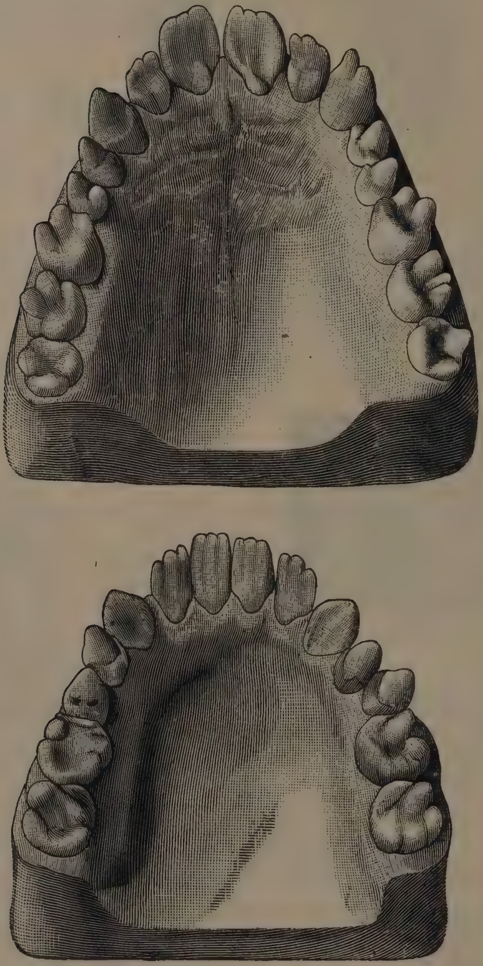

FIG. 44.

found in the first molar; but junction of the roots is more common in the second molar than in the first.

The third molar presents an increased number of cusps; the whole tooth may be reduced to a comparatively small size; the roots are often joined together and curved well backwards. In one variety the roots are joined together, but bifurcate towards the apices and present four small roots (fig. 42). A peculiar abnormality of the posterior external cusp is shown in fig. 43. Occasionally the roots are grooved or even canalised by the inferior dental nerve. (See fig. 49.)

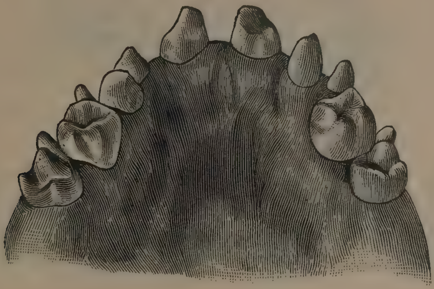

FIG. 45.

(3) Variations from the normal, involving many teeth.—A few cases of this character have been recorded. A case which occurred in the practice of C. Robbins[1] is shown in fig. 44. In two sisters of this patient a similar condition existed, though not so well marked, and some cousins were also stated to have exhibited the same peculiarities. The models are interesting, as they throw light upon the genesis of the cusps.

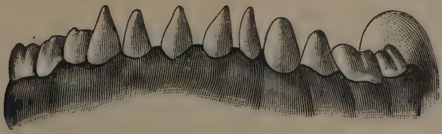

FIG. 46.

The remarkable abnormalities of teeth shown in figs. 45 to 47 were recorded[2] by H. Moon. The models were taken from the mouths of two sisters, one aged eleven (fig. 47) and the other aged fifteen (figs. 45 and 46). The hair of the younger sister was short, fine, and scanty. Her eyes were of a grey colour, remarkably small, the sight of the left eye being defective from birth. The hair on the scalp of the elder sister was short and scanty, but her temples and cheeks were thickly covered with very fine hairs.

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[1] *Trans. Odonto. Soc.*, xxiii, 80.

[2] *Ibid.*, ix, 232.

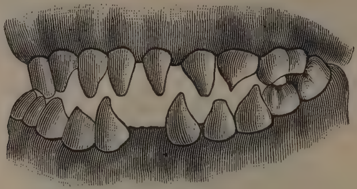


FIG. 47.

(D) VARIATIONS OF THE BLOOD-VESSELS OR NERVES.

Lepkowski[1] has shown that in rare instances there are, in the embryo, anastomoses between the pulp vessels and the vessels external to the tooth germ, and it seems likely that these anastomoses may occasionally persist and give rise to the abnormality shown in fig. 48, of a blood-vessel entering a tooth from its lateral aspect.

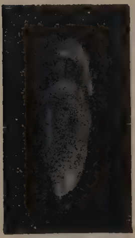
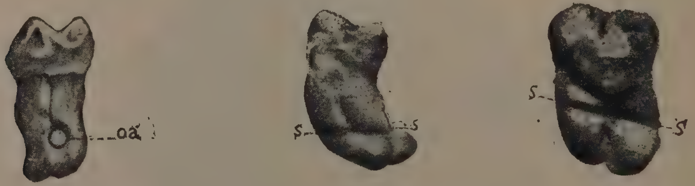


FIG. 48.

The root of the third molar may be grooved, or perforated by the mandibular vessels and/or nerve (fig. 49).

More rarely they perforate the root of the second molar; in a case in which this occurred, the third molar root was only grooved.[2] Perforation of the root of the second premolar may occur, but it is extremely rare.



Foramen through root. Groove on buccal surface. Groove on lingual surface.

FIG. 49. (From *Brit. Dent. Journ.*)

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[1] *Die Vierteilung der Gefässe in den Zähnen des Menschen* (Anatomische Hefte) H. liv.

[2] J. B. Parfitt, *Brit. Dent. Journ.*, February 15, 1921.

(E) VARIATIONS IN COLOUR.

It is not proposed to discuss at this point variations in colour caused by extrinsic factors such as stains, derived possibly from food substances, or metallic substances used either as filling materials or in the occupation of the individual; nor is it proposed here to discuss variations in colour due to changes in the calcification of the dental tissues, but those colour changes which are due to deposits in the dental tissues during the process of their development. Two such variations are known; neither is common.

Green teeth.—Several cases are on record in which, subsequent to prolonged or intense jaundice during the first weeks of life (icterus gravis neonatorum), the teeth calcifying at that time have been coloured green on eruption; the staining is caused by the deposition of bile pigment from the circulation during their calcification.

Histological examination[1] of these teeth has shown that the stain appears in definite bands of colour in the dentine of teeth calcifying at birth; and that the enamel has only a slight general stain, though that formed post-natally has a deeper stain.

Pink Teeth.—Teeth varying from pink to deep red or mahogany are found in cases of congenital hæmatoporphyrinuria (of Garrod), the colour being due to the deposition of pigment from the circulation during the calcification of the teeth. The pigmentation occurs in the dentine and cementum only, and is not found in the enamel; both deciduous and permanent teeth are affected. As other porphyrins are found in the urine besides hæmato-porphyrin the name *porphyrinuria* is now usually used for this condition, which is a congenital error of metabolism.

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[1] P. E. Boyle and M. Dinnerman, *Amer. Journ. Orthodont. Oral Surg.*, 1941, xxvii, 377.

CHAPTER III

Abnormalities in the Structure of the Teeth.

Abnormalities in Structure may be divided into :

(i) Those due to causes acting before calcification of the crown is complete.

(ii) Those occurring after calcification of the crown is complete.

Those of the second group are dealt with in the chapters treating of their causative factors.

With two exceptions (mottled teeth and those associated with some cases of osteogenesis imperfecta) all the abnormalities included in group (i) are coincident with abnormalities in form.

On account of the fact that a tooth calcifies progressively from its occlusal surface to its apices, markings caused by interferences with development as seen on the external aspect of a tooth are usually arranged transverse to the long axis of the tooth, and the position and extent of these markings informs one of the approximate age of the individual at and during which the etiological factor was active.

The calcification of a tooth is not a continuous process, but is characterised by short periods of activity followed by intervals of quiescence, so that if the etiological factor is active only during a period of quiescence, the teeth may escape any interference with their development.

In the past two different conditions have been taken as being present in what is often called 'hypoplasia'[1] of the enamel; they are :

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[1] *Hypoplasia* means *under-development*, and hence popularly *defective formation.* With reference to the abnormalities in structure discussed in this chapter the use of this word is not altogether a happy one ; for instance, some of these abnormalities are defective in form but not in calcification ; the converse is true of others, one is defective in neither and some vary ; similarly, a tooth may be normal in form and calcification but diminutive in size, *i.e.* under-developed and therefore hypoplasic—yet it is not usual to apply this term to diminutive teeth. The retention of this term in dental scientific nomenclature is therefore open to objection.

(*a*) Its irregular distribution or partial absence.
(*b*) Its deficient calcification.

Histological work repeatedly brings home the fact that quite often—indeed, in certain varieties it may be usually—each of these conditions when present exists separately; for instance, many teeth with marked defective distribution of enamel (condition (*a*)) are found histologically to have the enamel far more highly calcified than do apparently normal teeth of the same age, and many teeth where the enamel is apparently normal in bulk and distribution are histologically not so densely calcified as normally.

It is commonly thought that these characteristic markings are due to faulty deposition of calcium salts, but it is far more probable that they are caused by deficient enamel matrix being produced for the calcium salts to be deposited in, and that the calcification in such matrix as is produced takes place primarily in the usual manner. The contours of the brown striæ tend to confirm this opinion and lend weight to the view that deposition of calcium salts in the enamel matrix may take place via the pulp.

In addition, we have little or no knowledge of the variations in stability of the salts which go to the make-up of enamel, whether it is densely calcified or not.

The structural defects dealt with in this chapter are described as they occur when obvious to the naked eye, but in some cases the defects are in such minor degree that magnification may be required to see them; this has been termed 'microscopical hypoplasia.'

The abnormalities included under group (i) may be classified according to their etiology:

1. Local causes
    - (*a*) Trauma.
    - (*b*) Infection (local).
2. General causes
    - (*a*) Heredity.
    - (*b*) Infection (producing a specific variety of defect).
    - (*c*) Trophic disturbances.
    - (*d*) Chemical imbalance.
    - (*e*) Congenital causes.

The defect produced by trophic disturbances is by far the commonest variety.

(1) Abnormalities in Structure due to local causes.

These almost invariably affect one tooth only.

(*a*) **Trauma.**—The abnormalities in structure produced by this means are described in Chapter IX under the heading 'Traumatic injuries of the teeth'; they are known as dilacerations and also present abnormalities in form.

(*b*) **Infection.**—Inflammation and suppuration occurring in connection with deciduous teeth may interfere with the calcification of successional teeth; molars, being the most prone to caries of the deciduous teeth, most often cause this condition, and the mandibular second premolar is most often affected, though any permanent tooth may be affected by its deciduous predecessor.

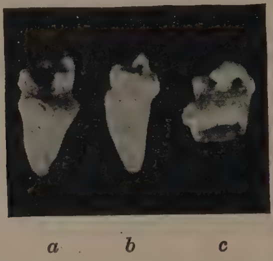

FIG. 50.—*a, b, c*: Local defects of mandibular premolars, specimens from different mouths, each being the only defective tooth in the dentition of which it formed a unit, and due to a diseased deciduous predecessor. (E. S.)

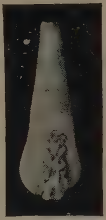

FIG. 51.—Maxillary permanent canine with markings of defective formation arranged vertically. (E. S.)

(From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

The appearance of the affected teeth differs widely (fig. 50) and depends on the degree of interference with their calcification and the stage of development reached when the interference took place. The enamel may be slightly pitted at the part forming when the interference took place, or it may be very faulty, discoloured, decreased in thickness, pitted and grooved, or even absent in parts; the dentine may contain many interglobular spaces, and the roots also may be stunted. Occasionally there is constriction at the part which was forming when the interference was most active, and the shape of the tooth sometimes indicates that the portion calcified before the interference took place was pushed to one side, so that on the completion of calcification a form of dilaceration is found. Sometimes calcification may be so interfered with that the apex never closes and the tooth ends in a widely expanded portion. (See also p. 291.)

Rarely the defectively formed part may be limited to one aspect of a tooth only (fig. 51), the junction of the normal and defective portions showing as a vertical line. Such condition may be caused when the etiological factor is strictly localised and of long standing.

Local structural defects very rarely occur in the deciduous dentition. Two cases have been recorded by A. T. Pitts; in one, a maxillary second molar failed fully to erupt, the crown was dwarfed and cone shaped; in the other, a maxillary first molar had the anterobuccal portion affected; there had been a sinus in the region of this tooth three weeks after birth, the tooth erupting at six months.

(2) Abnormalities in Structure due to general causes.

(*a*) **Hereditary defects.**—There are two forms of hereditary defect :

General hereditary defects.
The unerupted teeth in Cleido-Cranial dysostosis.

Neither is common.

General hereditary defective formation (**Hereditary Opalescent Dentine**) of the teeth is a rare condition. It differs considerably in degree, and in all cases there is a partial absence of the enamel ; in the mildest forms the enamel is well formed but shows numerous shallow pits. Both deciduous and permanent teeth are usually affected. In one case under observation this condition was present in two children, the father, and the grandmother.

With an increase in the deficiency of the enamel, the teeth may appear of a dark brown colour (fig. 52). A case of this character was recorded by S. Spokes.[1] There were grooves in the long axis of the tooth, and in some places there was a total absence of enamel, while in other places the enamel was present in irregular patches. The family history of the patient revealed the fact that a similar condition existed in many members of the family, and that the deciduous as well as the permanent dentitions had been affected. The character of the teeth was examined in forty-two individuals extending over four generations, of whom twenty-three had defective enamel.

An account of a family exhibiting this type of defect was recorded by J. G. Turner,[2] who was able to trace the condition through five generations. Of the fifty individuals composing the table, twenty-one showed the abnormality (fig. 53). The abnormal condition was always transmitted through abnormals. The defect took the form of a stunting of the teeth accompanied by marked defect of the enamel, both the deciduous and the permanent dentitions being affected. The number of teeth varied in the different individuals. The earlier formed teeth were most affected and in all the cases the third molars, when present, had a normal covering of enamel.

In extreme degrees of this condition (fig. 54) the dentine is unable to withstand the force of attrition and rapidly becomes worn down to the gum margin ; it then appears dark brown or amber yellow and semi-transparent. The reason for this latter

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[1] *Trans. Odonto. Soc.*, June 1890, 229.
[2] *Ibid.*, xxxix, 137.

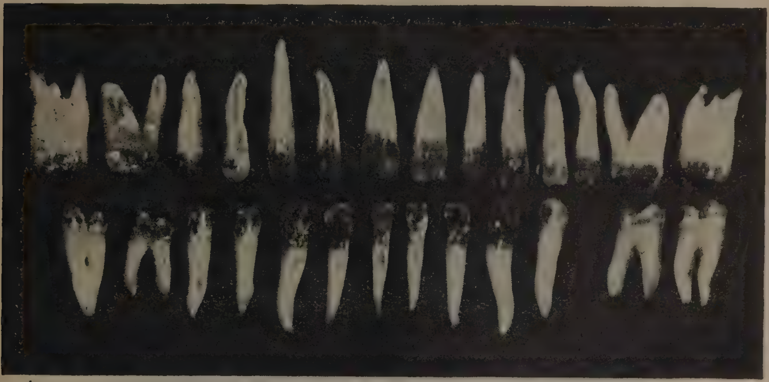
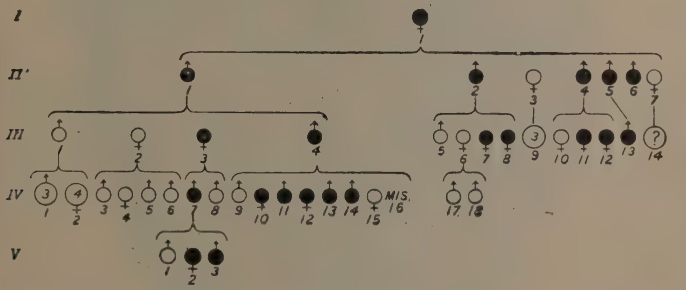


FIG. 52.—Teeth from a case of general hereditary defective formation.

appearance is explained by the histological findings. The section (fig. 55) is a mesial section made from a premolar from a



● indicates an affected person. ○ indicates an unaffected person.
A large circle indicates a childship. The figure inside indicates the number of children. Where sex is known it is added.

FIG. 53. (Turner.) (From *Trans. Odonto. Soc.*)

female aged seventeen, and shows that nearly all the dentine tubes are entirely occluded, and their contents calcified to such an extent (probably in response to thermal and other stimuli on the exposed dentine) that their refractive index has approximated that of the dentine matrix and so rendered the dentine very translucent, so that the tubular structure of the dentine is seen with difficulty, and only in the root portion does one find an occasional patent tubule ; in addition, the stimuli apparently caused the pulp to continue forming dentine without any break in its continuity, so that the pulp chamber in the coronal portion is entirely obliterated and almost so in the root. J. H. Mummery

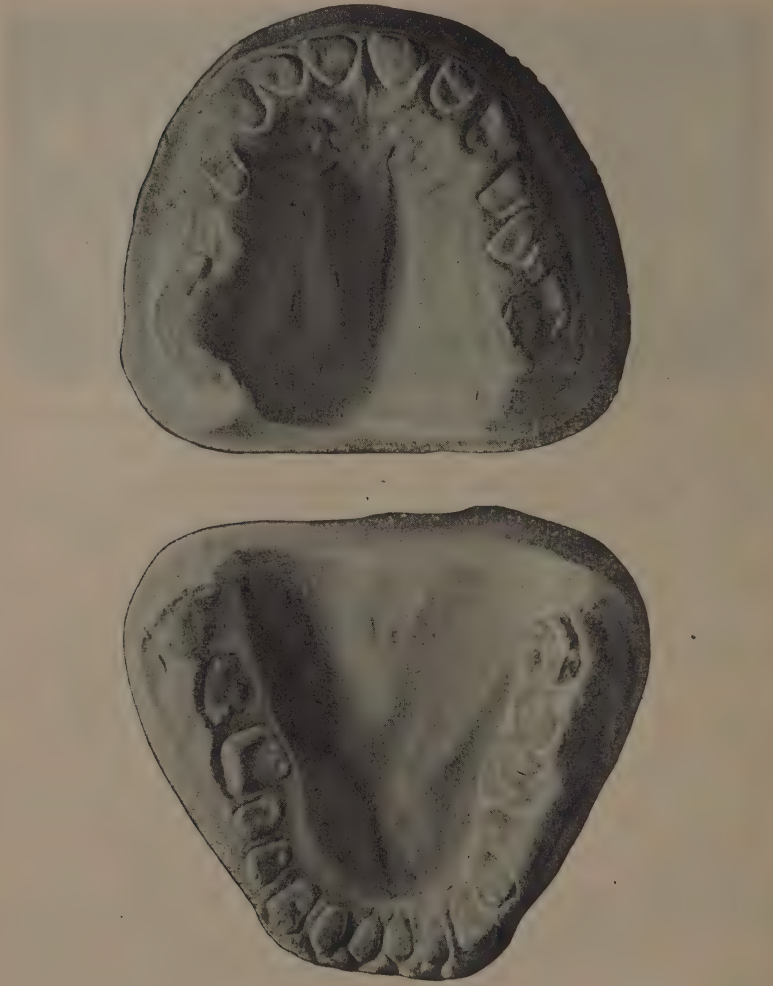


FIG. 54. (From the Royal Dental Hospital Reports, 1910.)

has recorded from the teeth illustrated in fig. 52, that the enamel structure was dense and hardly penetrable by fuchsin, which seems to imply that it was hypercalcified.

These teeth are much softer than normal when sections are ground for histological examination. According to Hodge[1] and others the bony skeletal system is normal in these cases, as also is the saliva with regard to its chemical and bacteriological content, and the only difference they can find in individuals possessing these teeth is the high plasma phosphatase value in the children; in the adults the values are 5 to 7, which is normal, but in children aged from eleven to sixteen the values range from 9·5 to 17·5, and in those aged from three to nine years from 18 to 25, or three times the normal for children. Curiously other children in the affected families but who possessed normal teeth also showed this abnormality.

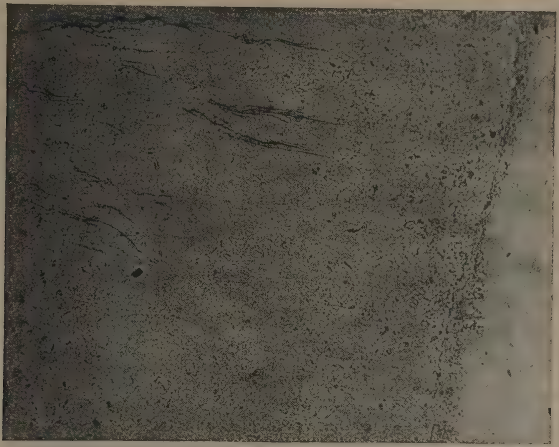


FIG. 55.—Photo-micrograph of a ground section of a mandibular premolar from a case of general hereditary defective formation. Female, aged seventeen years. The pulp-cavity is entirely obliterated except in a small portion of the root, the dentine is so highly calcified as to show hardly any trace of tubules and then only by careful focussing; those tubules seen, which are in the root, are almost the only ones remaining patent; the main mass of dentine appears almost structureless. (E. S.) (From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

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[1] See *Journ. of Heredity*, 1938, xxix, 359, and *Journ. Amer. Dent. Assoc.*, 1939, xxvi, 1663.

A high plasma phosphatase value also occurs in adult and infantile rickets, osteitis fibrosa and osteogenesis imperfecta, in all of which bone deficiencies of one form or another occur.

The defect occurs as a typical dominant and is not sex linked, and those possessing the defect when mated to normals transmit the defect to about half their children, as has been seen in the earlier record of cases on page 44.

The dentine is stated to have a high water and a low inorganic content, but the percentages of calcium and phosphorus normal; chemically it is said to resemble compact bone rather than normal dentine.

Such enamel as is present is also translucent and has a bluish or greyish appearance. The cementum is normal.

In many respects these teeth resemble those found in some cases of osteogenesis imperfecta (see p. 65) and the possibility occurs to one that their occurrence may be part of an incomplete syndrome of that disease.

It is possible that there are other variations of this condition, or other conditions in some respects similar, but unrelated to it; for instance the following two cases have been seen (E. S.): a girl of eighteen, with the full dentition for her age, having the enamel of all teeth freely pitted on all aspects, but no opalescence or abnormal attrition; and a boy of fourteen having markedly translucent teeth with bluish enamel, but the enamel normal in amount and distribution; no other abnormality could be elicited in either case. A high degree of translucency of enamel and dentine has also been seen in some, but not all, cases of osteitis fibrosa diffusa (hyperparathyroidism).

The unerupted teeth in **Cleido-cranial dysostosis**. This disease being hereditary presents another, but rare, form of hereditary defect in the unerupted teeth.

These teeth have been described by M. A. Rushton,[1] who suggests that they are compression forms, the compression being caused by the non-absorption of the bony crypt to allow for the proper growth of the contained tooth, so that the crowns tend towards a more conical or spheroidal shape and have the enamel ridged and pitted in places; the roots are not so evenly curved as in normal teeth and have sudden small changes in their direction (fig. 56). In these particular cases the condition is hereditary, but as Rushton points out, 'the abnormalities found in the unerupted teeth in the dysostosis are the same as those often seen in unerupted teeth, including supernumerary ones, in otherwise normal persons,' and suggests that the mechanism may be the same in both cases, though in the latter the bony abnormality may be local, and occasionally, though not necessarily, hereditary.

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[1] *Brit. Dent. Journ.*, 1937, lxiii, 641.

(*b*) **Infection** (producing a specific variety of defective formation).—**Congenital syphilitic infection** is the only infection known to produce a specific abnormality in the structure of the teeth.

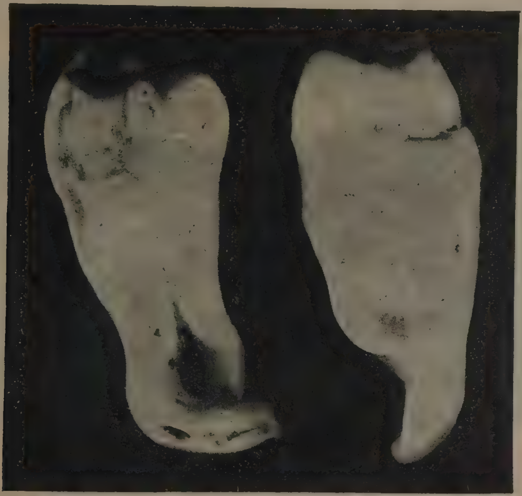

FIG. 56.—Unerupted premolars from a case of cleido-cranial dysostosis showing root distortion. The crown of the tooth on the right has a deep enamel defect and appears compressed when seen from the occlusal aspect. (M. A. Rushton.) (From *Brit. Dent. Journ.*)

The permanent teeth affected are usually the maxillary and mandibular incisors ; less frequently the first molars, occasionally the canines, and rarely the premolars.

Many congenital syphilitics have macroscopically normal teeth, but in cases where the teeth are typically affected the appearances are almost pathognomonic.

A few cases have been recorded in which the deciduous teeth are said to have been affected, but the authenticity for such statements makes them of doubtful value, and caries of the incisal edge may simulate the specific appearance ; probably they escape being affected because abortion usually results if the infecting agent is active when these teeth are calcifying.

The affected teeth are sometimes of a 'muddy' colour, but may be normal.

Syphilitic incisors ('Hutchinsonian incisors') are small barrel-shaped teeth and are usually spaced (fig. 57). The long axes of the maxillary first incisors often tend to converge towards their incisal edges, and the alveolar portion of the jaw in this region is often imperfectly developed. The teeth are generally affected symmetrically, but exceptions occur, for example, when one incisor is perfectly formed and the other presents the typical syphilitic form.

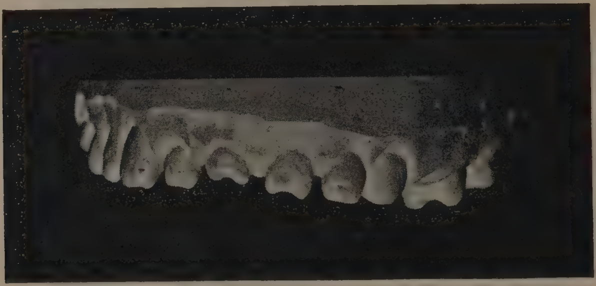

FIG. 57.—Model showing syphilitic maxillary first incisors. (G. G. Campion.)

On the eruption of a normal incisor tooth, the cutting edge is surmounted by three tubercles separated by two shallow notches; the tubercles rapidly become worn down by attrition, so that the cutting edge soon appears to be quite straight. In the syphilitic tooth the outer tubercles are well marked, but the central tubercle is badly developed or absent, and the effect of attrition is to produce one central notch between the two outside tubercles. The second incisors are not always affected by syphilis, the first incisors being considered by Jonathan Hutchinson as the 'test' teeth.

The deformity of syphilitic canines shows itself by a circumferential notch near the cutting edge or point of the crown. Syphilitic molars, like the first incisors, are smaller than normal. They are dome-shaped, and the crown surface is of an irregular pattern, instead of having well-developed cusps (see fig. 58). They are sometimes known as 'Moon's molars.'

The premolars, which are only rarely affected, are smaller than normal and have a stunted and domed appearance with little or no enamel at the tips of the cusps ; the domed appearance is comparable to that of the permanent first molar.

The *Treponema pallida* is to be found in the dental follicles in proximity to the vessels and their walls. Fig. 59 is from a paper by Pasini.[1]

Cavallaro, in an exhaustive paper on the Relation of Syphilis to Dentition,[2] at first denied, but later (1925) confirmed the discovery of Pasini that the *Treponema pallida* is to be found in the dental follicle. He shows that a curious modification of the lower ends of growing teeth can often be demonstrated in the form of a constriction of the dental papilla which clinically corresponds to the atrophy of the tooth cusps so often seen. He states that at this part certain changes can be noted in the pulp tissue, the nuclei are better stained, and the infiltration of the embryonal cellular elements is more intense. He also has found in the dental follicles the following alterations—namely, endovasculitis, perivasculitis, and hæmorrhage. In addition to the changes in the follicle already referred to, he finds certain histochemical alterations in the enamel and dentine.

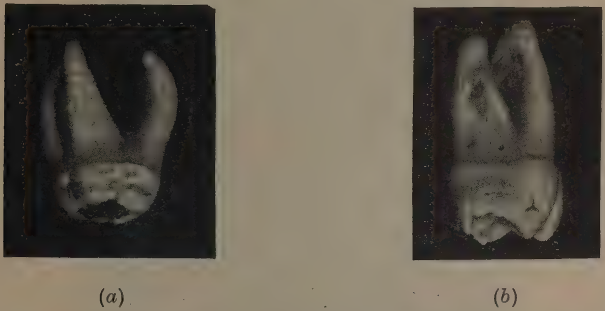
(*a*) (*b*)

FIG. 58.—The syphilitic molar (*a*) is shown in contrast with a normal-shaped molar (*b*)—both slightly enlarged. There was a typical history of congenital syphilis. (G. G. Campion.)

Hill[3] examined many sections of developing teeth from nine congenital syphilitic full-term fœtuses, but was unable to find the organism ; possibly the fact that many congenitally syphilitic children do not have the teeth affected may account for this, though he found it in the livers of six of them.

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[1] *La Stomatologia*, November 1908, 5.

[2] *Dental Cosmos*, November and December 1908 ; January and February 1909.

[3] *American Journ. of Path.*, September 1931.

Cruikshank [1] suggests that the maldevelopment of the central tubercle of the Hutchinsonian incisor is consequent on the specific vascular obstruction, and found that of all congenitally syphilitic children 28 per cent. show the dental stigmata in these teeth, though Nabarro found only 15 per cent. and Hissard under 6 per cent. The notching when present is demonstrable by X-ray before eruption. Mandibular first incisors showed the stigmata in 5 per cent., canines in 4·5 per cent. and permanent first molars in 7 per cent. of all cases.

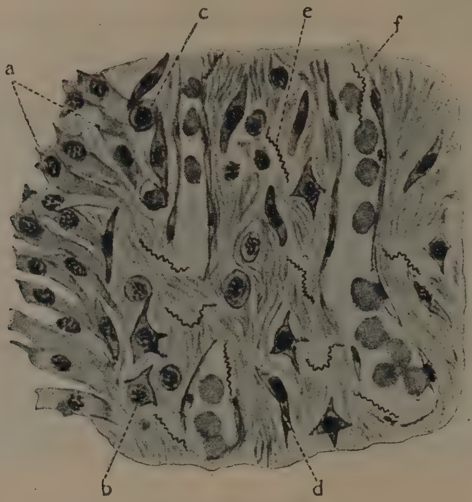

Fig. 59.

(*a*) Odontoblasts of irregular shape and distribution.
(*b*) Polygonal cells } of the dental papilla.
(*c*) Round cells } of the dental papilla.
(*d*) Fusiform cells } of the dental papilla.
(*e*) *Treponema pallida* situated in the gelatinous tissue of the dental papilla.
(*f*) *Treponema pallida* situated in the lumen of a capillary and partly attached to the endothelium.

If anti-syphilitic treatment is carried out very shortly after birth no dental stigmata develop, but if from age one to two upwards they may appear.

According to Davis [2] untreated syphilitic mothers bear about 20 per cent. normal non-syphilitic children, the remaining 80 per cent. being represented by abortions, stillbirths and living syphilitic babies.

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[1] L. G. Cruikshank, *Proc. Roy. Soc. Med.*, 1938–39, xxxii, 343.

[2] M. L. Davis, *Archives of Pedriatics*, 1938, lv, 590.

If syphilis is recognised and treatment started before the fifth month of pregnancy fœtal infection can nearly always be prevented, and even when treatment is started later the incidence is considerably reduced. It seems probable that the younger placenta is impervious to the passage of spirochætes and that it is the erosive changes about the middle of pregnancy that make it permeable to them.

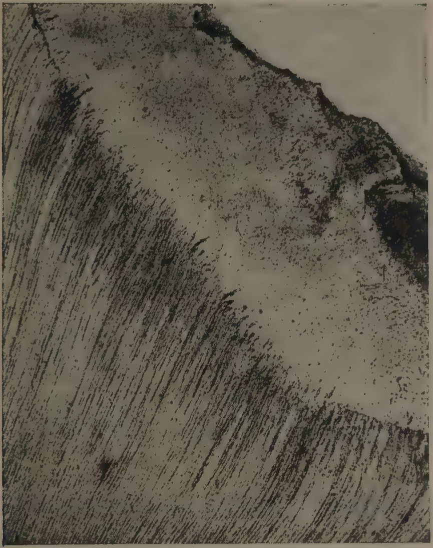

Fig. 60.—Photo-micrograph of ground section of maxillary permanent first molar affected by congenital syphilis, showing partial obliteration of the normal amelo-dentinal junction by the brush-like protrusion of the dentinal tubules into the enamel. A part of this section only showed a trace of interglobular spaces, which are not here seen. (E. S.) (From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

It has been said that syphilitic teeth are poor in structure and therefore readily attacked by caries or quickly worn by mastication. Many patients, however, seen with characteristically syphilitic teeth do not confirm these statements.

Histologically, Leon Williams has found that the enamel is very faulty, and that the dentine contained interglobular spaces in abundance; one's own findings by no means confirm this (fig. 60), interglobular spaces may hardly be present at all, nor does there seem to be much difference in the structure of the enamel though it is less in amount, but the amelo-dentinal junction is far from normal and may present an appearance of dentine fibrils forming brush-like terminations, so masking the line of junction of the two tissues.

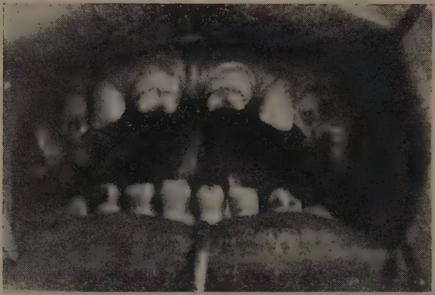

Fig. 61. (London Hospital Dental Museum.) (E. S.)

Occasionally a defect due to trophic disturbances may be superimposed on a syphilitic defect. The writer has seen two such cases (fig. 61).

The diagnosis of syphilitic teeth.—Syphilitic incisors must be diagnosed from (*a*) trophic defects in incisors, where the deformity is confined to the edge and the central portion wears down more rapidly than the side. (*b*) A notch not infrequently seen on the incisal teeth of women, which is caused by the sudden impact of the teeth when cotton, etc., is bitten through instead of being cut. The teeth, however, are otherwise normal in shape. (*c*) Developmental notches, apart from the depressions normally occurring between the three or more tubercles normally seen on the cutting edges of incisor teeth, may also occur (fig. 62). The

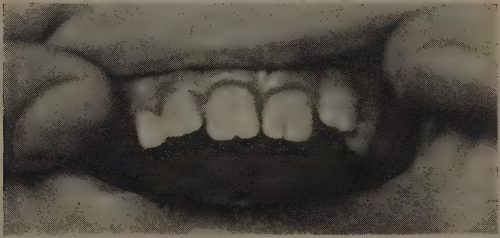

Fig. 62.—Female, age 7, showing developmental (non-specific) notches on the incisal edges of the four maxillary incisors. (E. S.)

notch produced upon the teeth by the use of a clay pipe ought not to be mistaken for a syphilitic notch, as it is altogether different in character and always involves at least three teeth, two in one jaw and one in the other (fig. 63). The diagnosis of congenital syphilis can also be confirmed by the presence of other lesions, such as scars radiating from the angles of the mouth (rhagades), dusky-coloured skin, Parrott's nodes on the forehead, broad depressed bridge of nose, interstitial keratitis, and patches of alopecia ; the Wassermann and Kahn reactions should always be tested if doubt exists, though their accuracy is doubtful after the first decade.

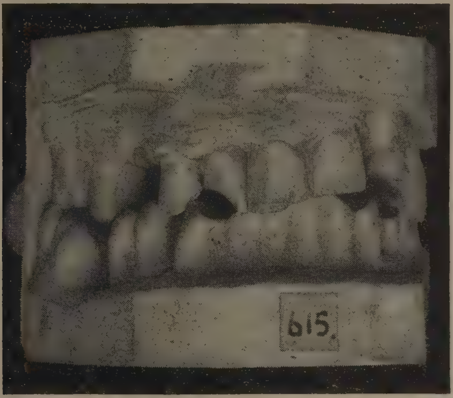

FIG. 63.—Abrasions formed by clay pipe smoking ; that on the patient's right involved four teeth. There were other similar abrasions on the left side in front of and behind the maxillary canine. The patient's occupation was selling clay pipes and he advertised his wares by holding several in his teeth simultaneously. (E. S.)

It is well to remember that the diagnosis of congenital syphilis by no means rests upon the presence or absence of these teeth, for they are only met with in a small proportion of cases. H. Moon has suggested an explanation of the deformity. He believes that 'the peculiar shape results from a stunted development of the first formed portion of dentine—in other words, a dwarfing of the cusps ; and that the single central notch on their cutting edge is due to a greater diminution in the size of the central than the lateral lobes.'

Pflüger[1] and others have suggested that the characteristic shapes of syphilitic teeth may be due partly to their being compression forms, brought about by inflammatory processes in the surrounding tissues, the slow calcification of the predentine, and the non-absorption of their bony crypts to allow for the proper growth of the contained tooth germs, and this view is supported by their occasional later eruption.

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[1] H. Pflüger, *Dtsch. Zahn. Mund. Kieferh.*, 1936, 3, 293.

The lesions on the teeth caused by syphilis are usually found on the incisors and first molars of the permanent series, and this is to be attributed to the fact that the maximum intensity of the virulence is during the last months of intra-uterine life and the first three months of extra-uterine life, which is the period when the affected portions of those teeth are forming. Welander[1] reports a case of a lad who acquired syphilis when between three and four months of age from his nurse. In this patient, the teeth characteristic of syphilis were present. The second and third molars have not been recorded as being affected by congenital syphilis.

It is not uncommon to find that there is deficient bony development in the anterior maxillary region, so that a condition of anterior open bite exists.

(*c*) **Trophic disturbances.**—The great majority of cases of structural defects in teeth are caused by this means ; it causes it in both the deciduous and permanent dentitions. It is more convenient to deal with them as they occur in the permanent dentition first.

Permanent dentition.—Defects due to trophic disturbances during the calcification of the teeth are common among all grades of society, but their incidence varies among different groups. Among children who are well cared for the incidence is as low as 4 to 5 per cent., among others it may rise to as high as 15 per cent., but the incidence and the severity of its effects appear to be getting less as the increased knowledge of the nutritional requirements of very young children is put into practice.

By trophic disturbances is meant any cause apart from those covered by the other headings in this classification, which may interfere with the normal growth of the individual. Such causes arise from a variety of factors and are mostly transient, their period of activity varying from a few weeks to several years. These causes are :

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[1] *Nord. Med. Archiv*, xxvii, No. 3.

(i) Food deficiencies.
(ii) General infection.

Defects due to trophic disturbances mostly affect those portions of teeth which are calcifying during the earliest years of life, and the cause, unless it is a very transient one, may affect the development of the individual as a whole. It is therefore the food deficiencies and illnesses of those earliest years which concern us in so far as they may affect the structure of teeth, though occasionally one finds that portions of teeth are affected which do not calcify till five years or even more have passed.

(i) *Food deficiencies.*—The ideal food for the first nine months or more of a child's life is that which nature provides, and the adequacy of that secretion depends in turn on efficient maternal feeding. Many children, however, for one reason or another, are hand-fed ; some, on milk foods made to simulate their natural food ; others, on foods containing an excess of starchy and farinaceous material. Sometimes these artificial foods are not readily assimilable and so give rise to gastro-enteritis. The digestive capabilities of children vary considerably, and a diet which one child assimilates readily will cause gastro-enteritis in another. Besides this, other not readily assimilable substances may be present in the artificial food or there may be an absence of necessary chemicals or vitamins, or both, so that, even if no digestive trouble occurs, there is a deficiency of growth-producing factors which may be reflected in deficient formation and calcification of the teeth.

Such interference with the metabolism and development of the child if prolonged may produce scurvy or rickets, though other factors, such as absence of sunlight (in particular), fresh air, and exercise are ancillary to the production of this latter disease. It may be thought that rickets does not occur so early in life as to affect those portions of the teeth which are commonly marked ; Preston Maxwell,[1] however, has shown that inadequate diet during pregnancy may result in fœtal rickets and that the deciduous teeth in such cases may be defective on eruption after birth ; the cause may be present from the earliest years, but not perhaps in sufficient intensity to produce the typical diagnostic signs of rickets for several years, after which its cumulative effects may be more readily seen. Any or all such deficiencies and disturbances may cause trophic defects, of different degree, according to their severity, and of different extent according to their duration.

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[1] *Proc. Roy. Soc. Med.*, xxviii, 265.

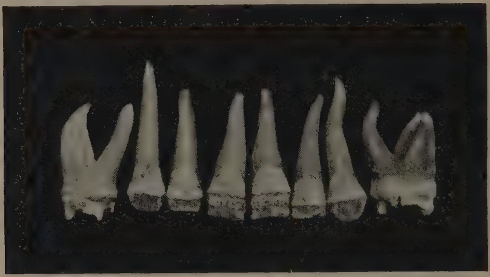

FIG. 64.—Defective maxillary incisors, canines, and first molars from the mouth of a patient who had suffered from rickets.

Coincident with interference with the formation and calcification of the teeth is interference with the calcification of bone, osteoid tissue in some measure replacing bone.

(ii) *General infection.*—Any infective disease occurring while the calcification of the teeth is in progress may interfere with their normal development. Epidemic gastro-enteritis (summer diarrhœa) until recently was one of the commonest infections to which children were subject in the first twelve months of life, and there can be but little doubt that it was a common cause of trophic defects.

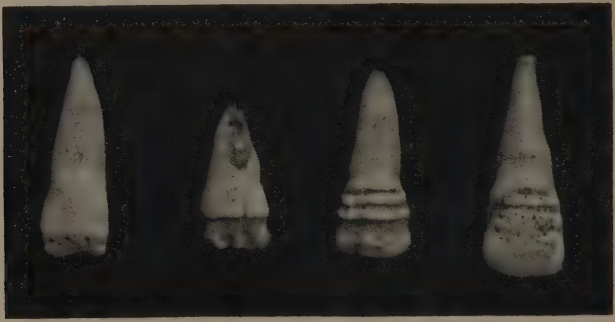

FIG. 65.—Examples of defective maxillary first incisors.

Acute infective fevers, such as scarlet fever, measles, or diphtheria, are well-recognised causes, and any other acute infective illness must also be regarded as a possible cause.

The teeth involved (figs. 64, 65, 66, 67).—The permanent incisors, canines, and first molars are the teeth usually involved in severe cases. Occasionally the premolars are involved as well, or they alone may be involved; rarely the second and still more rarely the third molars have been involved.

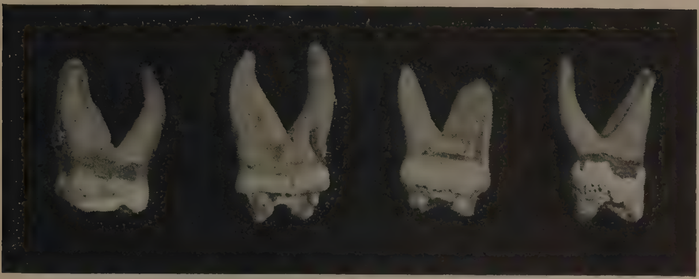

FIG. 66.—Examples of defective maxillary first molars.

The extent to which the teeth are involved.—Those parts of the teeth which are calcifying during the period of trophic disturbance are the parts affected, and the degree to which they are affected is commensurate with the severity of the disturbance.

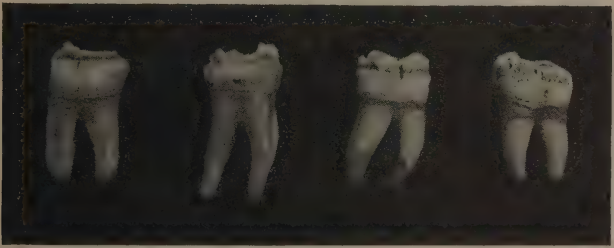

FIG. 67.—Examples of defective mandibular first molars.

The macroscopical appearances vary according to the extent and degree of defect present. In mild cases, the surface of the enamel presents opaque white patches intermingled with normal enamel, producing a mottled appearance which often becomes unevenly stained later on. In more severe cases there is pitting and grooving of the enamel of varying depth and extent, or the enamel may even be absent in places and the surface of the crown irregular, rough, and pigmented; incisal cutting edges may be uneven and jagged, and the cusps of the first permanent molars sharp and spiky.

In defects caused by short acute illnesses a single row of pits or an encircling groove may be found transverse to the long axis of an otherwise normal crown. The occurrence of several such rows or grooves may be caused either by separate acute short illnesses, or by exacerbations or other illnesses occurring during a prolonged trophic disturbance such as rickets. An acute short illness does not necessarily cause a defective marking on a tooth, as the illness may have occurred during one of the periods of quiescence in its calcification. One very remarkable, constant, and so far unexplained fact concerning this variety of defect is that the portion of enamel at the neck of the tooth is always apparently normal.

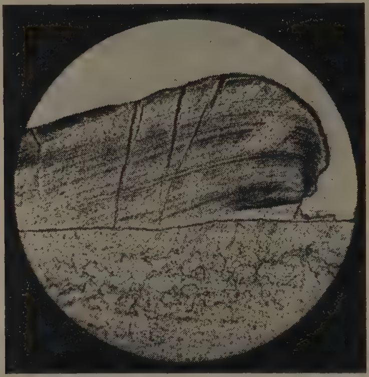

FIG. 68. (E. S.)

Histologically the enamel is thinner than normal and laid on unevenly; in severe cases it may even be absent over pits and grooves, and there it shows a rounded contour where it rather suddenly fades to absence; the brown striæ of Retzius follow such abnormal contour instead of their usual sweeping curve; and so are irregular and may even coalesce in places (fig. 68).

Normally, the brown striæ are not parallel to either surface of the enamel, but as they represent what was once the external surface of the forming enamel, they bear a definite relation to it.

Striation of the enamel prisms is said to be more marked than normal, but though this may sometimes be the case it is by no means always so and, indeed, frequently the reverse occurs. Similarly the brown striæ are said to be more marked, and though this sometimes is so it is by no means constant.

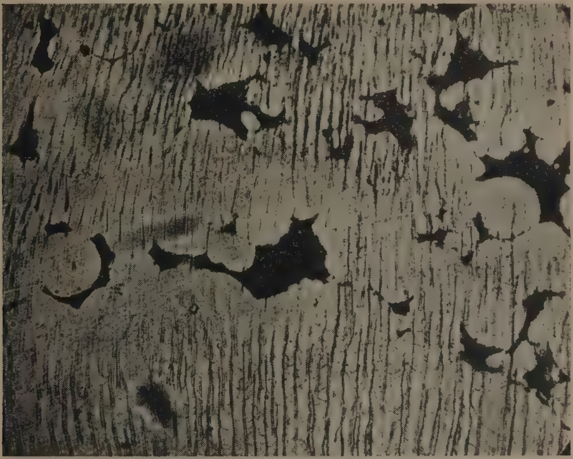

Fig. 69.—'Hypoplasic' dentine permeated with silver nitrate. The tubules and their branches immediately around each 'space' appear not so permeable as elsewhere. (E. S.)

Nasmyth's membrane has been described as being thickened in the pits and crevices; J. G. Turner considers this to be in part due to the presence of uncalcified or semi-calcified enamel, but it is probably also (or alternatively) due to its being well protected from the wear of attrition at those points. Experimental permeation of such enamel by stains and silver salts from the pulp aspect demonstrates that it is often not so permeable via the interprismatic substance as enamel from apparently normal teeth of the same age; this indicates that though the enamel is small in amount and uneven in distribution it is more highly calcified than normal.

The dentine shows interglobular[1] spaces, relatively few in a mild case but of wide extent in severe cases, adjacent to, but never at, the amelodentinal junction ; when the cause has acted intermittently they are sometimes arranged in rows (fig. 70), which indicate the extent of calcification which had taken place when the causative factor came into action.

Deciduous dentition.—Trophic defects occur in the deciduous dentition, but are rarer. Preston Maxwell[2] in a valuable paper on adult and fœtal rickets has shown that inadequate diet during pregnancy may result in fœtal rickets, and that the deciduous teeth when they erupt may be defective. It is only logical to infer that other chemical or vitamin deficiencies in the maternal diet may have a like result. Kingston Barton found the incidence to be about 2·5 per cent. and occurring in hand-fed children only ; he examined 202 children, 67 of whom were hand-fed. A. T. Pitts, examining 4,000 children, found it in 15 cases only, or 0·37 per cent. Usually all the deciduous teeth are involved, but chiefly the canines and second molars, which one would expect to be affected more by post-partum conditions as they are developed a little later than the others. The causes are similar to those causing trophic defects in the permanent teeth, and may in the first place act through the mother particularly during the later months of intra-uterine life.

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[1] The incidence, reason for, and position of interglobular spaces is of great interest and, at the moment, by no means understood. They are regarded by some as being pathological, and to the extent that they are increased in trophic disturbances this impression is certainly created, but the facts that they only rarely occur in the roots of teeth (save in cases of dilaceration) and never in contact with the amelodentinal junction in spite of severe experimental trophic disturbance (*vide* M. Mellanby's various papers), do not bear this out. Moreover, the fact that they occur normally in some marsupials, cheiroptera, ungulates, cetacea, and possibly other mammalian orders, seems to indicate that their incidence in man may be similar ; they seem to occur universally in young human teeth in civilised communities, but become obliterated as age and wear advance, though sometimes a sign remains that they have been present. Histological work on such dentine (in man) seems to indicate that they may be an alternative means of nourishment to the dentine, as the dentine tubes and their branches immediately surrounding such spaces are almost or quite impermeable to diffused reagents, so that there is, apparently, an area of hypercalcification immediately around interglobular spaces (fig. 69).

The fact that the position of the spaces under ordinary circumstances is so constant, *i.e.* $\frac{1}{8}$ to $\frac{1}{10}$ of the distance from the amelodentinal junction to the pulp chamber, in other mammals besides man, seems to indicate their normality. In either—or any—case, if they were due to trophic disturbance their position should vary so as to affect only that portion of tooth which was calcifying when such disturbance took place, but it does not.

[2] *Proc. Roy. Soc. Med.*, xxviii, 265.

Other associated conditions.—Lamellar cataract is frequently associated with defective teeth. N. G. Bennett found the tooth abnormality associated with twenty-two out of twenty-six cases of lamellar cataract.

The Birth Line (Neonatal Line).—Rushton [1] has pointed out that in sections of deciduous teeth there is a definite contour line which indicates the junction of the prenatally and postnatally formed enamel, and has named it the birth line. From later investigation [2] of sections placed between crossed Nicol prisms he concludes: ‘that during the neonatal arrest of growth calcification of prisms proceeds to a high degree, but calcification of interprismatic substance is low or is of an unusual kind, and that this “fault” in the line is not afterwards repaired.’ He

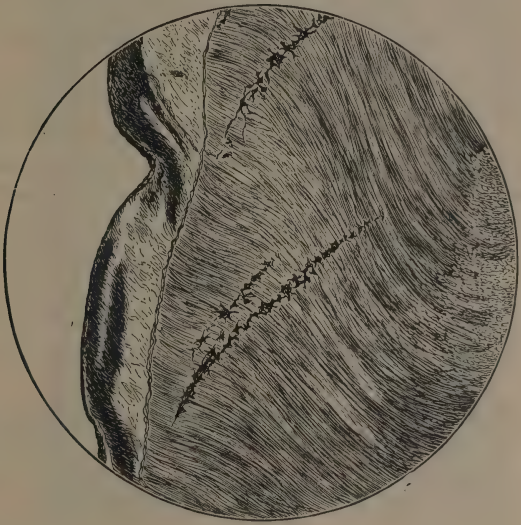

FIG. 70.

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[1] *Dental Record*, 1933, liii, 170.

[2] *Brit. Dent. Journ.*, 1939, lxvii, 1.

also shows that other and more externally placed lines may be found indicating the approximate dates of illnesses or periods of impaired health.

Kronfeld and Schour [1] have shown that a neonatal line may also be seen in the dentine, though the line in the enamel appears to be the more obvious. They also showed that in cases of birth injury the line in the enamel may be emphasised.

It seems clear that these lines indicate a break or interference in the continuity of calcification, probably of a trophic nature, and so may be regarded as a microscopical form of ‘ hypoplasia,’ especially when emphasised by injury at birth, or, as in the case of later lines, by illnesses.

The Teeth in Scurvy.—There is a form of structural defect of trophic origin which occurs in dentine and is due to deficiency or absence of vitamin C. It is not yet clear to what extent, or how, such deficiency may affect calcification of the teeth of man, though it is well known that young children may suffer from scurvy and relatively commonly from sub-scurvy. As these effects are only produced during the formative stage they are best seen in rapidly growing teeth of persistent growth, and so have been studied intensively in guinea-pigs, animals particularly responsive to absence or deficiency of this vitamin.

In conditions of scurvy Höjer [2] describes the earliest change in the teeth of this animal as being in the odontoblast layer, these cells become shorter, more rounded, and lose their regular layer arrangement by tending to curve inwards towards the centre of the pulp ; the layer is then split by the cells secreting a hard substance between themselves, and the odontoblasts are then transformed into or replaced by osteoblasts in bony canals. There is also a widening of the dentinal tubes in the dentine formed before the commencement of scurvy.

Instead of new dentine forming new bone is formed internal to the already existing dentine, and this, if the animal does not die but is kept alive by subnormal doses of vitamin C, may permeate the whole pulp. (Figs. 71 and 72.) There is an early hyperæmia of the pulp, and sometimes there are hæmorrhages.

The new pulp-bone is of a spongy, porous character, and its formation is followed by atrophy and resorption of the pulp tissue.

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[1] *Journ. Amer. Dent. Assoc.*, 1939, xxvi, 18.

[2] *Acta Pædiatrica*, Uppsala, 1924, Supp. iii, 38.

If the vitamin dosage is increased to the extent of the healing of the scurvy lesions, the pulp-bone becomes reorganised into irregular dentine, osteodentine, with bone canals and dentinal canals. (Fig. 73.)

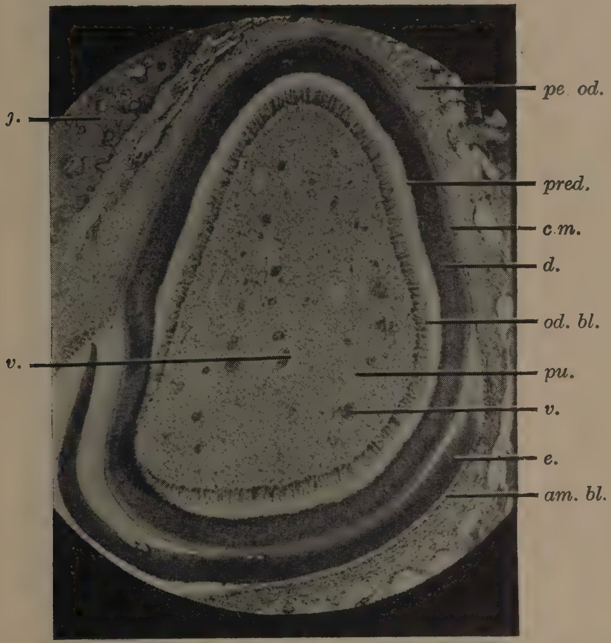

FIG. 71.—Cross section of normal mandibular incisor of guinea-pig. *j.*, jaw bone; *pe. od.*, periodontium; *c.m.*, cement membrane; *d.*, dentine; *pred.*, predentine; *od. bl.*, odontoblasts; *pu.*, pulp; *am. bl.*, ameloblasts; *v.*, blood vessels; *e.*, enamel. (From 'Studies in Scurvy,' J. Axel Höjer, *Acta Pædiatrica,* Vol. III (Supplement), Uppsala, 1924.).

In sub-scurvy—the form which is more common in man—the effects are similar but not so pronounced, and the changes do not deviate so far from the normal.

(*d*) **Chemical Imbalance—Mottled teeth.**—This condition has been fully described by F. S. McKay and G. V. Black. The defect of the enamel consists of ‘ minute white flecks or yellow or brown spots or areas, scattered irregularly or streaked over the surface of the tooth, or it may be a condition in which the entire tooth surface is of a dead paper-white like the colour of a china dish. In many cases the surface of the tooth is dotted with irregular, shallow pits, which are usually darkly discoloured because of the lodgment of debris. Any or all of the teeth may be mottled or certain groups only ; or the area of some teeth either above or below a certain point may be mottled, and the rest of the enamel of such teeth be practically normal.’

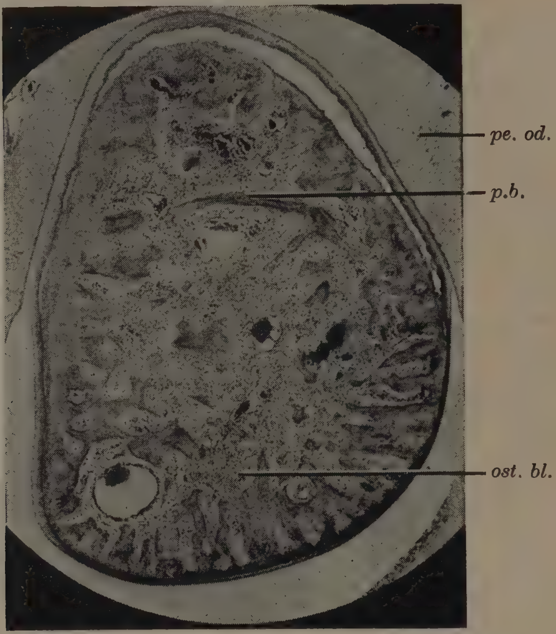

FIG. 72.—Well-marked scurvy, 46 days. Cross section of mandibular incisor of guinea-pig. Pulp-bone (*p.b.*) fills the pulp ; *ost. bl.*, osteoblasts ; *pe. od.*, periodontium. (From ‘ Studies in Scurvy,’ J. Axel Höjer, *Acta Pædiatrica.*)

The essential malformation is a failure of the cementing substance between the enamel rods on the outer one-fourth to one-third of the surface of the enamel. In about 40 per cent. of the cases a colouring matter—named by Black 'brownin'—is formed in place of the cementing substance. This discoloration is not present when the teeth erupt, but gradually makes its appearance and is usually located on the labial aspects of the anterior maxillary teeth.[1]

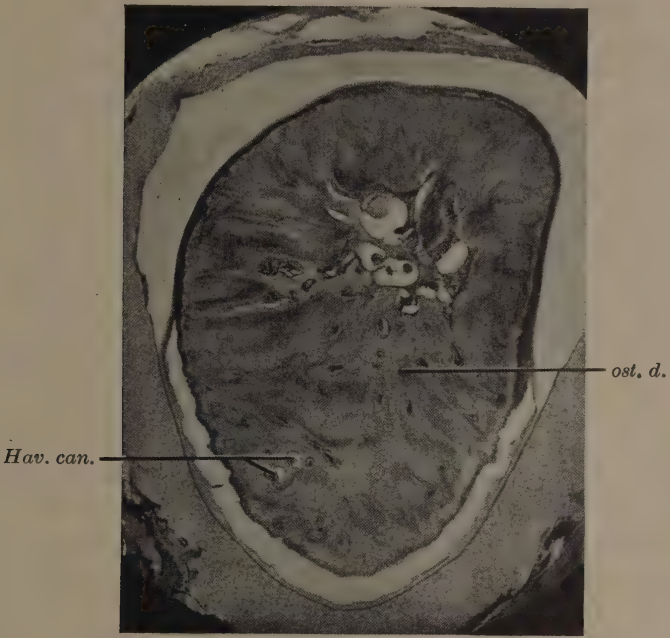

FIG. 73.—Healing scurvy. Cross section of mandibular incisor of guinea-pig. Osteodentine (*ost. d.*) fills the pulp. *Hav. can.*, bone canals. (From 'Studies in Scurvy,' J. Axel Höjer, *Acta Pædiatrica.*)

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[1] Normally there appears to be a greater permeability (tubularity) of the enamel of very newly erupted human teeth from the outside for about $\frac{1}{6}$ or a trifle more of its thickness; the permeability of this area soon becomes uneven and patchy, and eventually the area becomes hypercalcified; it is in this portion apparently that the colouring matter in 'mottled teeth' is deposited. All evidence at the moment is that the extra deposition of calcium salts in this area after eruption takes place from the pulp, unless such interprismatic area is of the nature found in mottled teeth, when it appears to be penetrated by stains from the outside. A similar area is found in young marsupial teeth and, very noticeably, in the enamels of many fish.—E. S.

This defect of the enamel was met with in certain well-defined areas in the Rocky Mountains, and occurred only in those individuals who were born in any of these areas or who had lived there continuously during the period of enamel formation. A similar condition is found in Braintree and other localities in Essex and also in parts of Somerset and other parts of the United Kingdom. It is now said to occur in many districts in the United States and in parts of Italy. The writer has seen this mottling affecting every tooth in the permanent dentition of a young woman born and bred in Braintree, though its usual distribution is as described by McKay and Black.

In a paper by N. J. Ainsworth[1] on the frequency and distribution of this condition at Maldon, in Essex, and the surrounding district, it was found that there was an excess of fluorine in the water supply of the localities where it occurred, and this writer otherwise confirmed the findings of McKay and Black. Following on this Bowes and Murray,[2] working on Maldon teeth, state that typical mottled teeth can occur when the fluorine in the water is two parts per million, and that Maldon water contains five parts per million; and that mildly mottled teeth from a fluorine area do not differ much in composition from non-mottled teeth, but that the quantity of fluorine is definitely greater than in the controls, there being half as much again in the enamel and three times as much in the dentine. The results of their analyses are as follows :—

RESULTS OF ANALYSIS. COMPOSITION OF MALDON ENAMEL AND DENTINE COMPARISON WITH CONTROL TEETH.

	Enamel		Dentine	
	Mottled	Non-mottled	Mottled	Non-mottled
Nitrogen ...	0·3035 per cent.	0·156 per cent.	—	—
Calcium ...	37·01 ,,	37·07 ,,	28·4 per cent.	27·79 per cent.
Phosphorus	17·24 ,,	17·22 ,,	13·58 ,,	13·81 ,,
CO_2 ...	2·347 ,,	1·952 ,,	3·798 ,,	3·176 ,,
CO_2 after ashing ...	0·55 ,,	0·54 ,,	Nil	Nil
Magnesium	0·371 ,,	0·464 ,,	0·907 ,,	0·835 ,,
Fluorine ...	0·032 ,,	0·025 ,,	0·073 ,,	0·025 ,,
Ca/P ...	2·146 ,,	2·153 ,,	2·091 ,,	2·012 ,,
Ca/CO_2 ...	15·77 ,,	18·99 ,,	7·48 ,,	8·75 ,,
P/CO_2 ...	7·345 ,,	8·8 ,,	3·576 ,,	4·15 ,,
Ca : P : CO_2 (molar)	10 : 6·04 : 0·56	10 : 5·99 : 0·48	10 : 6·17 : 1·22	10 : 6·12 : 1·04

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[1] *Brit. Dent. Journ.*, September 1933.

[2] *Ibid.*, 1936, lx, 556.

By analogy with what takes place in the teeth of rats fed with sodium fluoride they conclude that the possible cause of the whiteness, which is present before discoloration, is lack of, or abnormal distribution of, iron in the enamel.

According to T. G. Ni[1] mottled teeth occur in those parts of China, notably Chekiang, where fluoride deposits occur; this author states that 'the fluorine content of foodstuffs grown near a fluorite mining area, with some exceptions, do not seem to have been influenced greatly by the fluorine content of the soil, which varied from 100 to 500 parts per million.' However, the fluorine content in Chinese tea from a fluorite area varied from 15 to 1757·8 mg. per kilogram of dry substance, whereas that from ordinary soil varied from 37·5 to 178·8, and Indian and Ceylon tea from 8·7 to 38·1 mg. per kilogram. There is also a high fluoride content in salted Chinese vegetables which comes from the salt used, the fluorine content of which varies in China from 0·8 to 28·5 parts per million.

It is, however, a fact that sporadic cases of mottled teeth occur in districts where excess of fluorine is not known to occur. It appears that the absence of some of the interprismatic substance in the external portion of the enamel is the essential factor, and that whether it is caused by excess of fluorine or not the brown stain or mottling will ultimately occur there, just as it often occurs in exposed dentine.

It is possible that some cases of mottled teeth, when affecting the whole dentition, may be a minor degree or variety of hereditary defect.

(*e*) **Congenital Causes—Osteogenesis Imperfecta** (formerly Fragilites Ossium).—This disease has been described as existing in two forms, *congenita* which makes itself evident in early life, and *tarda* a later form; they are believed, however, to be really the same disease, congenital in each case, and the time of onset of signs and symptoms immaterial in its nomenclature. The teeth in some cases of this disease present abnormalities in structure, but not in form. Five teeth were removed from a boy aged ten suffering from this disease; he had had fourteen fractures of bones in the lower limbs, a brother was stated to have had six or seven fractures of his femora and a sister two or three fractures, also in lower limbs; he had not blue sclerotics. Both the deciduous and permanent teeth were very translucent throughout their whole extent, more so than are the teeth in severe General Hereditary Defective Formation. They were yellowish-grey in colour, and the pulps could be seen throughout their extent; they appeared to be of a remarkably even degree of calcification, and the amount and distribution of the enamel to be normal. Excess of calcium salts had been given over a prolonged period preceding the extraction of the teeth. There was some dental caries present.

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[1] *Journ. of Clin. Med.*, 1937, ii, No. 3.

Such a dental condition is not common to all cases of this disease, and, indeed, may be rare and possibly dependent on the periodicity of the causative factor.

The crowns of the teeth from an adult case examined by Rushton [1] appeared dirty pinkish and opaque, and the teeth on section showed traces of what had probably been vascular channels in the dentine, the pulps were very small and the dentine showed but little trace of tubules and was as a whole so evenly calcified as to appear translucent and pale amber coloured in the roots; they were softer than normal. The dentine matrix was inadequately calcified and most of the dentinal tubules occluded and obliterated; there was no granular layer. The enamel showed minor imperfections but was not otherwise remarkable.

The teeth of the cases described seem to bear some resemblance to those found in the General Hereditary Defective Formation described on page 43, save that in the latter the enamel is defective in that it is pitted, grooved or even extensively absent in parts.

'Compression' Forms.—These are similar in description to the unerupted teeth found in cases of cleido-cranial dysostosis (see p. 46). It occurs in unerupted teeth, including supernumerary ones, and the bony condition believed to cause it appears sometimes to be hereditary.

Partial agenesis of enamel.—This is a rare condition. Two cases have been recorded in recent literature,[2] and we know of four others. It affects all the teeth in both deciduous and permanent dentitions. On eruption the amount of the enamel is as usual, but the superficial portion, especially on the occlusal aspects, is very defective, so that it can readily be scraped away, in some cases with a finger-nail, and this portion is soon worn or rubbed away (figs. 74 and 75). Clinically the teeth are coloured light brown or orange brown. Histological examination of teeth from two cases, one deciduous and one permanent (fig. 76), showed such enamel as remained to be particularly well calcified and with hardly a trace of brown striæ, and in the dentine no trace of interglobular spaces; in one portion of the enamel on a deciduous tooth there appeared a well-marked neonatal line which on the occlusal aspect only had separated the well-formed ante-natal enamel from the post-natal enamel which had been washed or rubbed away. The clinical appearance of these teeth is disfiguring. It is difficult to conceive what the cause may be, as it appears to affect the enamel of the crowns of all the teeth to a similar degree, and not that forming at any particular time or period. At present there is no evidence of its being hereditary.

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[1] Martin Rushton, *Journ. of Path. and Bact.*, 1939, xlviii, 591.

[2] *Proc. Roy. Soc. Med.*, 1936, 1542.

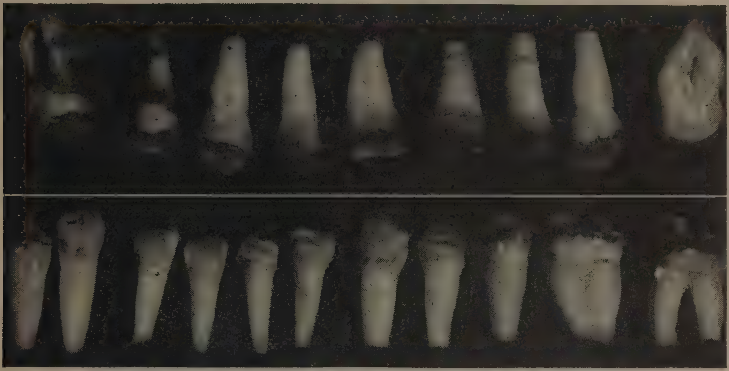

FIG. 74.—Permanent teeth from a case of partial agenesis of enamel, male aged 16. (G. B. Pritchard.)

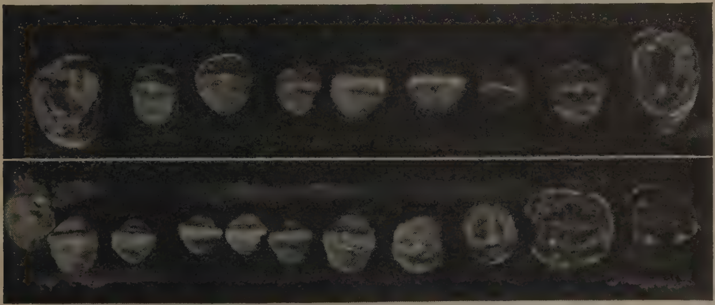

FIG. 75.—Occlusal aspect of the teeth shown in fig. 74. (G. B. Pritchard.)

A Rare Form of Dental Dysplasia described by Rushton[1] is that of a man of twenty-eight in perfect health, some of whose teeth became loose; the crowns of the teeth were normal in colour and shape; radiologically (fig. 76A) none of the teeth showed more than a trace of a pulp cavity and the roots were very short, many of them showed bone absorption at the apices sometimes having the appearance of dental cysts, most of the teeth showed one or more horizontal radiolucent lines, usually near the base of the crown. Microscopically the linings of the rarefied areas resembled the walls of ordinary dental cysts, the enamel was normal, the dentine of the whole upper part of the crowns was also normal, but rootwards from this point appeared to incorporate in its substance large numbers of pulp stones (fig. 76B). The radiolucent lines seen in the radiographs corresponded to the junction between the normal dentine of the crown and the rest of the tooth or to successive layers of pulp stones. The abnormality is related to the age or state of development of the teeth, and not that of the individual, and may also include abnormality of the sheath of Hertwig.

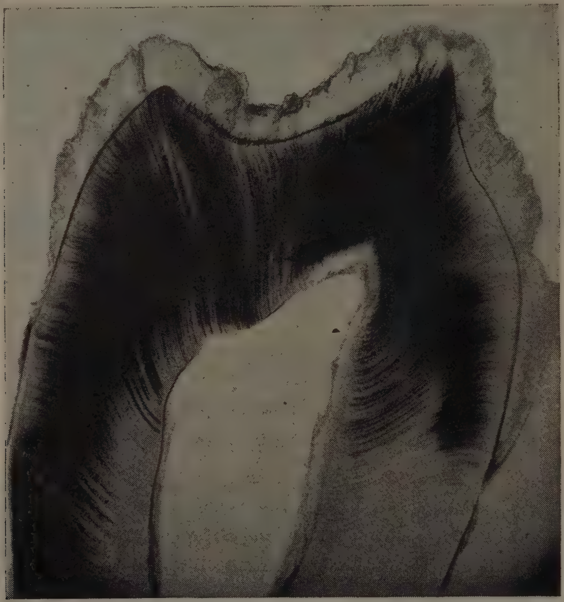

FIG. 76.—Ground section of a tooth from fig. 74. (G. B. Pritchard.)

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[1] *Brit. Dent. Journ.*, 1940, lxix, 165.

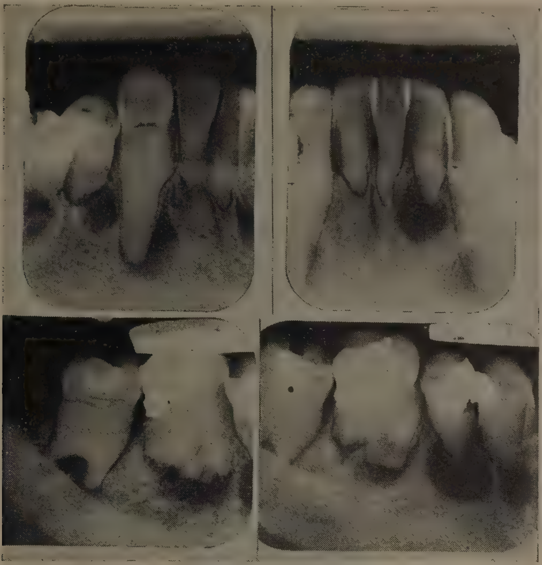

FIG. 76A. (M. A. Rushton.) (From *Brit. Dent. Journ.*)

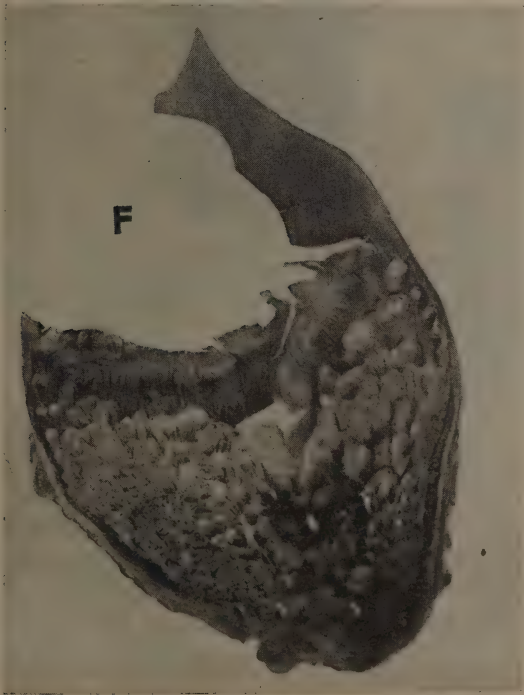

FIG. 76B.—Decalcified section. ⌈5 stained picrothionin. F, space occupied by filling. (J. F. Dudley.) (From *Brit. Dent. Journ.*)

INCIDENCE OF STRUCTURAL DEFECTS IN MAMMALS OTHER THAN MAN.

Investigation by one of us (J. F. C.)[1] has shown that similar defects in structure occur in the teeth of other mammals, both in those which have developed in the wild state, and still more commonly in those brought up in captivity. The developmental history of adult wild animals is of course unknown, but as far as can be judged the causes of the different forms of defect occurring in animals are similar or comparable to those affecting the teeth of man.

Thus **local defects** as the result of trauma have been found in such animals as polar bears, baboons and gluttons (fig. 77). The injuries in some cases are of the nature of dilacerations, accompanied, as is usual, with a local defect of formation at the point of injury, and sometimes, probably as the result of persisting local inflammation and infection, affecting some part of the tooth formed subsequently.

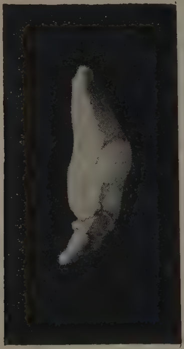

FIG. 77.—Local defective formation, baboon's maxillary right permanent second incisor. (From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

Conditions comparable to the **trophic defects** of man occur in baboons, other primates and carnivores both in a captive and wild state, but more commonly in the former (fig. 78). In the primates it appears that the maxillary permanent first incisors are particularly susceptible (fig. 79) and may be the only teeth affected.

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[1] 'Variations and Diseases of the Teeth of Animals,' 1936, Ch. XVIII.

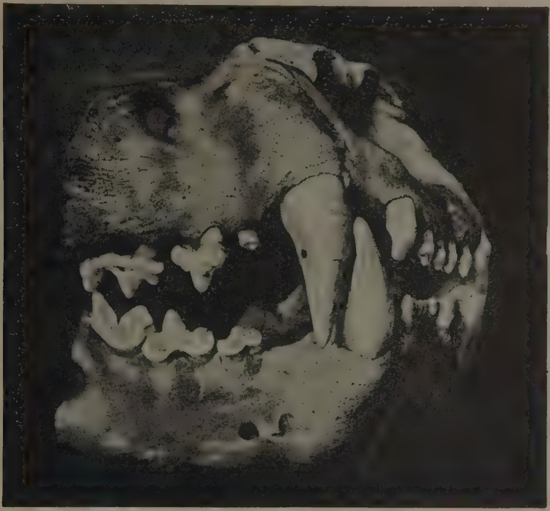

Fig. 78.—Skull of an Abyssinian lion about five years old. The canines, from the tips to about half-way up the crowns, are practically devoid of enamel, and the incisors, premolars, and molars all show defective enamel in places. (From *Trans. Odonto. Soc.*)

Even when it occurs in animals in the wild state, the etiological factor appears sometimes to be selective in its effect and involves some teeth more than, or to the exclusion of, others developing at the same time (fig. 80), though it is not always so.

Hereditary defects are almost impossible to prove in a wild animal, but the condition as it occurs in its milder degrees in man is occasionally simulated, and has been found in some six species (fig. 81). In any case it is difficult to conceive how else such a universal faulty development of the enamel of all teeth possessed by the animal could have arisen.

Mottled teeth also have been recorded in cattle, sheep and horses, and in a wild primate from Uganda, the cause apparently being the same as that which similarly affects the teeth of man.

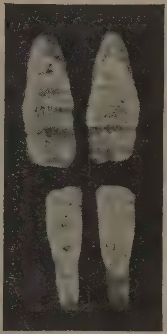

Fig. 79.—Permanent first incisors of a captive 'Black Ape' showing defective formation of the enamel. (From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

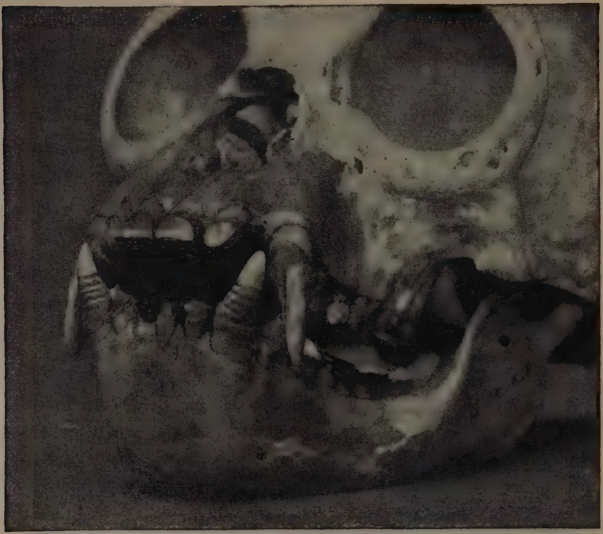

Fig. 80.—Hylobates concolor gabriellæ (Gabriel's Gibbon), B.M. 8.11.1.1. A specimen from the wild state showing several grooves on the mandibular canines and first premolars. (From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

For further information on the comparative incidence of these different forms of abnormality the student should consult the work of reference from which the above facts are taken.

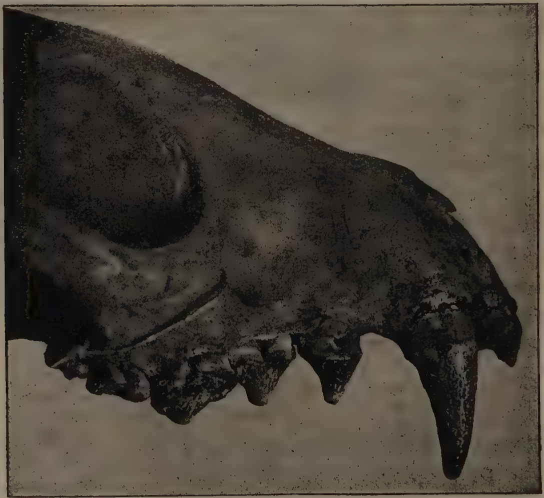

Fig. 81.—Macrogalidia musschenbroeki (Musschenbroek's Palm Civet), B.M. 98.11.10.1. The enamel of the teeth shows a condition simulating general hereditary defective formation. (From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

CHAPTER IV

Abnormalities in Position of the Teeth.

General Considerations—Classification.

(A) GENERAL CONSIDERATIONS.

(1) NORMAL ARRANGEMENT OF THE TEETH.

Before discussing the abnormal positions in which teeth may be found it is necessary to determine what are to be regarded as their normal positions.

In this connection it is to be borne in mind that the arches formed by the teeth vary to a considerable extent both racially and individually.

(*a*) **The Deciduous Teeth.**—If the mouth of a well-developed child of the age of five be examined, it will be noticed that the maxillary and mandibular teeth are arranged in arches with regular curves. The maxillary teeth form an arch which is greater than the arch of the mandibular teeth, and consequently they overlap the mandibular teeth on occlusion. On a close examination of the occlusal arrangement it will be seen that the maxillary first and second incisors overlap the mandibular first and second incisors in addition to the anterior half of the canine; the maxillary canine overlaps the posterior aspect of the mandibular canine and the anterior third of the first molar; the maxillary first molar occludes with the posterior two-thirds of the mandibular first molar and the anterior third of the second molar; the maxillary second molar occludes with the posterior two-thirds of the mandibular second molar and projects a little beyond it.

(*b*) **The Permanent Teeth.** — In the English race the usual arrangement is as follows: The canines and incisors form the arc of a circle; the premolars and the first and second molars pass back from the canines in straight lines diverging slightly outwards ; the third molar is placed a little internal to the arch. Variations from this arrangement are not uncommon ; for example, the premolars and molars may be arranged on a well-marked curve or in a perfectly straight line diverging outwards only very slightly.

The arch formed by the maxillary teeth is greater than that formed by the mandibular. The maxillary first and second incisors overlap the mandibular first and second incisors, the maxillary second incisor also overlapping the anterior half of the mandibular canine ; the maxillary canine overlaps the distal half of the canine and the anterior half of the mandibular first premolar ; the maxillary first premolar occludes with the posterior aspect of the mandibular first premolar and the anterior aspect of the second premolar ; the maxillary second premolar occludes with the posterior half of the mandibular second premolar and the anterior third of the mandibular first molar ; the maxillary first molar occludes with the posterior two-thirds of the mandibular first molar and the anterior third of the second molar ; the maxillary second molar occludes with the posterior two-thirds of the mandibular second molar and the anterior third of the third molar ; the maxillary third molar meets the posterior two-thirds of the mandibular third molar.

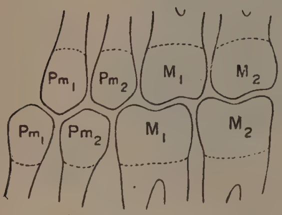

FIG. 82.

Normal occlusion.

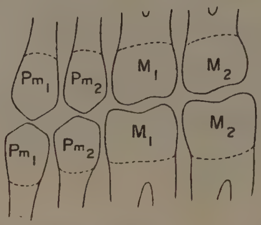

FIG. 83.

Half an occlusal unit post-normal.

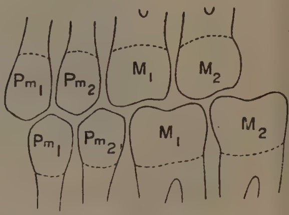

FIG. 84.

One occlusal unit post-normal.

(From *Proc. Roy. Soc. Med.*)

Any deviation from the arrangement described above must be regarded as abnormal, but before dealing with the causes of abnormalities of the teeth it is necessary to review briefly certain points in connection with the development and growth of the jaws and arches.

(2) DEVELOPMENT AND GROWTH OF THE JAWS.

The maxilla, formed from the maxillary process, arises as a membrane bone external to the cartilaginous nasal capsule and in the neighbourhood of the canine tooth germ by one centre of ossification. From this centre three processes rapidly radiate :—

(i) A nasal process which grows upwards.

(ii) An alveolar process which grows downwards, is thickened at its root to form the malar process, and might be called the alveolo-malar process.

(iii) A palatine process which grows inwards.

The premaxilla is formed from the meso-nasal process and fuses with the maxilla about the third month, the maxilla overlapping and almost excluding it from the face.

The mandible is developed in membrane as a single element, and its growth is determined to a great extent by that of the maxilla. At birth, the mandible consists of two halves joined by fibro-cartilage, and at about the sixth month the two segments unite. It is possible that the segments grow to some extent before they unite.

In the **maxilla** at birth, the tooth germs lie in close proximity to the orbital plate, the sinus being represented by a slight depression internal to and just above the follicle of the deciduous second molar. The crypts of the incisors and canines are complete, but between the first and second molars the septum is still imperfect. The second incisor lies slightly posterior to the first incisor, and the crypt of the first molar is in close proximity to it, the canine lying between and anterior. As the time for the eruption of the incisors approaches, the space between the second incisor and the first molar increases, the growth of bone at this point continuing until the eruption of the canine. There is reason to suppose that room is made in the arch for the canine by growth of bone at the suture between the premaxilla and the maxilla.[1] With the eruption of the second molars the deciduous dentition is complete. An examination of the mouth at this stage will show an interval between the second molar and the end of the alveolar ridge. This interval increases in size until the advent of the permanent first molar. A skull examined at this period, namely, about six years of age, would show the following condition of the permanent teeth: In the **maxilla** the second incisors are placed slightly posterior to the first incisors and their direction is more vertical. The premolars are embraced by the roots of the deciduous molars and their crowns are directed inwards, the second more than the first. The first premolar is normally situated close to the second incisor. The canine lies above and external to the arch of the incisors and premolars and is directed slightly outwards. The permanent first molar will be in the process of erupting and the occluding surface will be directed outwards to a slight extent. The permanent second molar is situated high up in the tuberosity of the bone, with its occluding surface directed downwards, outwards, and well backwards. In the **mandible** the second incisors lie in a plane posterior to the first incisors, and the canines are placed near the lower border of the bone and lie in a plane anterior to the second incisors with a slight tilt towards the median line. The premolars are embraced by the roots of the deciduous molars and their crowns are directed inwards. The permanent first molars are directed upwards and forwards, the second being under the base of the coronoid process with the occluding surface directed upwards, forwards, and slightly inwards.

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[1] See ‘Abnormalities in Position of the Teeth in Man,’ *Dental Record*, February 1914.

During childhood there is a progressive increase in the size of the arch, and in this way space is obtained for the permanent teeth, which are broader than the deciduous teeth, to assume a regular arch. The increase in the size of the arch can be demonstrated by taking models at regular intervals. The diagrams (figs. 85 and 86) were made from a series of models of two children of normal growth. The measurements were taken between the canines and the deciduous second molars with an instrument by means of which tenths of a millimetre could be registered. In the diagrams ten millimetres equal one millimetre of actual increase.

The diagrams show (1) that the principal increase in the intercanine width takes place between the ages of six to eight years, a period corresponding to the eruption of the permanent incisors; (2) that the increase in width between the deciduous second molars is gradual in character. The slight decrease in width between maxillary canines (fig. 86) was due to an inward displacement of the deciduous tooth by the erupting tooth.

The manner in which these changes in the arch are brought about is not clear. They have usually been attributed to a forward pressure exerted by the developing teeth, but this explanation is hardly satisfactory in view of the fact that the permanent canine lies above the deciduous canine and the premolars are embraced by the roots of the deciduous molars. There is some evidence that growth continues at the suture between the premaxilla and the maxilla during the first years of life, and A. Keith is of the opinion that growth also occurs along the median suture of the maxilla.

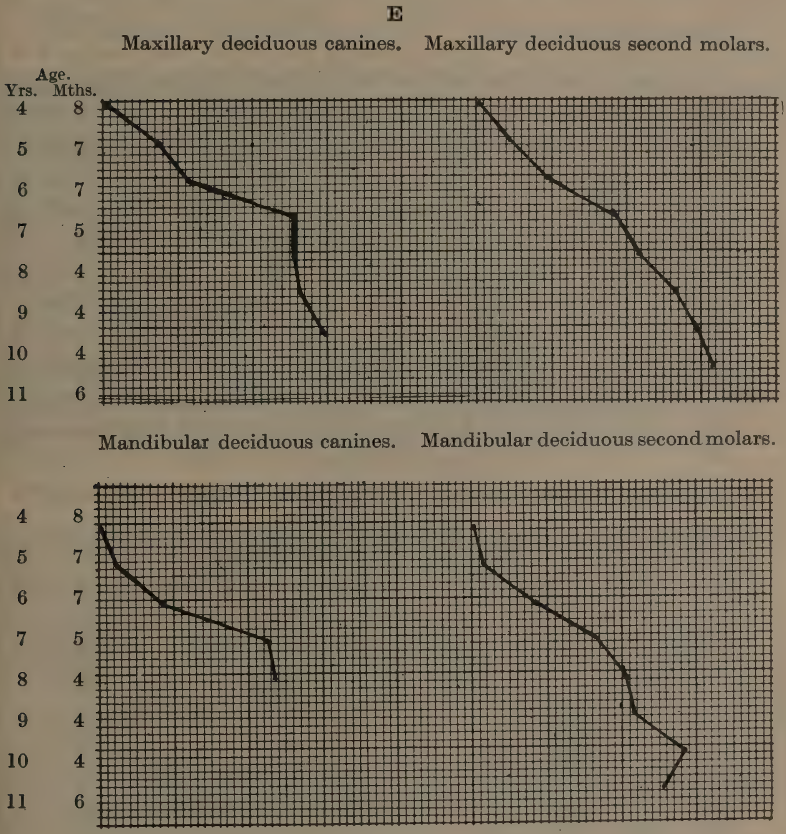

FIG. 85. (From *Dental Record.*)

The growth of the maxilla backwards provides room for the permanent molars. The growth of the maxilla in this region has been shown by Keith [1] to be dependent on the expansion of the maxillary sinus. At birth the position of the sinus is marked by a slight depression just above the follicle of the deciduous second molar. This depression spreads towards the orbital plate, and, at the end of the first year, has developed forwards over the deciduous first molar and backwards over the permanent first molar, an extension of the sinus rapidly taking place as the remaining molars are formed.

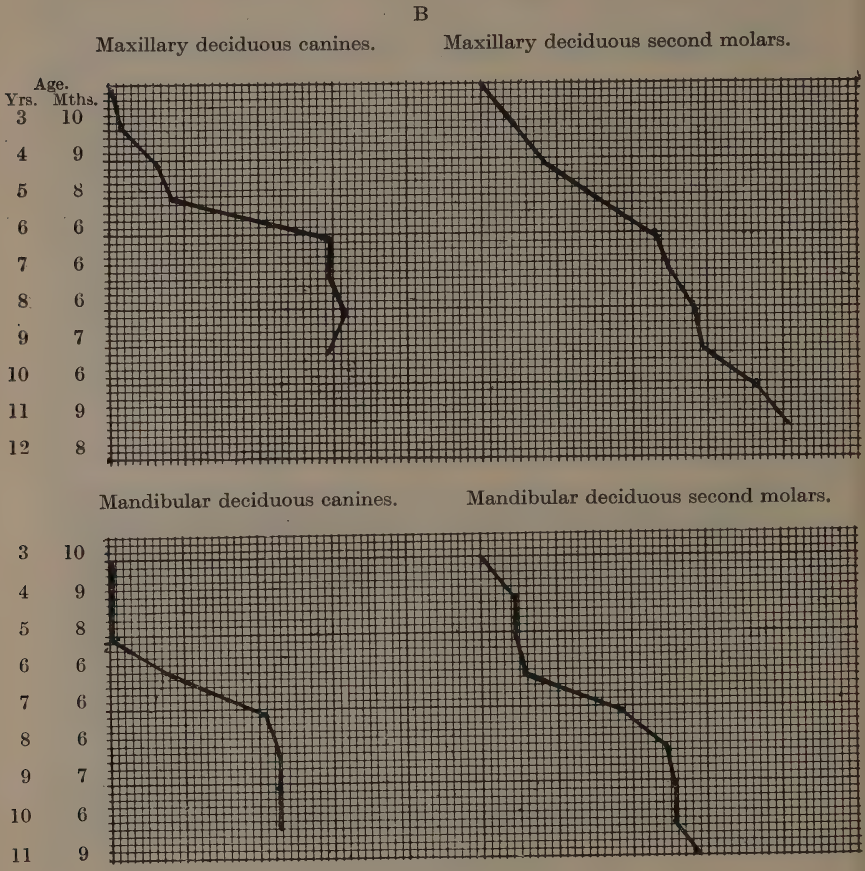

Fig. 86. (From *Dental Record.*)

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[1] A. Keith, *Brit. Journ. Dent. Sci.*, June 16, 1902.

The effect of the growth of the maxillary sinus on the eruption of the teeth can be studied by examining the relation of the teeth to the sinus in a sixth-year skull. The permanent first molar lies in close relationship to the sinus, with the occluding surfaces facing downwards and backwards. 'As the tooth moves into position, it rotates so as to bring its crown downwards and backwards, while the sinus spreads downwards and backwards between the roots of the teeth. The growth of the sinus wheels, as it were, the tooth into position. An exactly similar condition occurs with the second and third molars.'

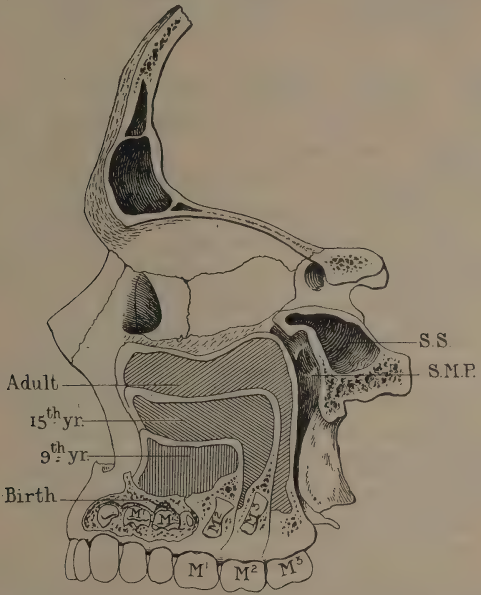

Fig. 87.—The above drawing by A. Keith illustrates the position of the developing molars and the relation of the maxilla to the pterygoid processes. 'The diagram is drawn to scale and is founded on material in the museums of the Royal College of Surgeons and of the London Hospital. Four stages are shown: (1) at birth, (2) at the ninth year, (3) at the fifteenth year, and (4) in the adult. . . . While the diagram demonstrates clearly the growth changes which occur in the jaw, it must not be forgotten that, in the body, the postero-superior border of the sinus is the fixed, and not the moving point as represented in the diagram.'

'The fulcrum on which this rotation of the bone takes place is formed by the body of the sphenoid, the anterior surface of its great wing, and the pterygoid plates, mainly the internal.' (See fig. 87.)

In the mandible the replacement of the deciduous teeth by the permanent teeth takes place in the same manner as in the maxilla, the space for the molars being obtained by a backward growth of the bone, this growth being correlated with the growth of the maxilla consequent on the expansion of the sinus. A recognition of these phenomena is necessary in studying the pathology of certain types of irregularities of the teeth.

In a paper, which students are recommended to study,[1] A. Keith and G. G. Campion have shown that the growth of the jaws is intimately correlated with the growth of the whole apparatus of mastication. The general nature of the growth-machinery involved is discussed with the view of showing the direction in which research is required. This paper lays stress on the close connection between irregularities of the teeth and other changes in the face and cranium.

The methods pursued were as follows :—

Measurements were taken to show growth in a sagittal plane, a line between the external auditory meati being taken as the base. From this base, measurements were made to the nasion E, the edge of the maxillary incisors F, and the mental eminence G. The measurements were carried out in individuals of four age periods : 3 years and 6 months ; 9 to 11 years ; 15 to 16 years, and adults. The growth in height was obtained by measuring from the nasion to the sub-nasal point (A), the maxillary incisors (B) and the sub-mental point (C) ; the growth in height of the front part of the mandible was obtained by measuring a line drawn from the top of the mandibular incisors to the sub-mental point (D). These lines of measurement are shown in fig. 88.

The region in which growth occurs can be ascertained by superimposing sagittal mesial sections of skulls of different ages, as shown in fig. 89. The skulls are superimposed so that pituitary fossæ and cribriform plates correspond. It will be noticed that the line E crosses three important sites, viz. fronto-nasal, spheno-ethmoidal junction, spheno-occipital junction. By superimposing these different elements as shown in fig. 90 an idea can be obtained of the relative growth in these situations. The authors found that in line E, the amount of growth in the fronto-nasal region was a superficial deposit ; at the fronto-ethmoid junction it was restricted ; but marked at the spheno-ethmoidal junction. This latter junction is concerned in providing space for the erupting molars, a subject already referred to on pp. 75 to 77.

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[1] *Dental Record*, February 1, 1922.

The growth sites crossed by the line F are the posterior area

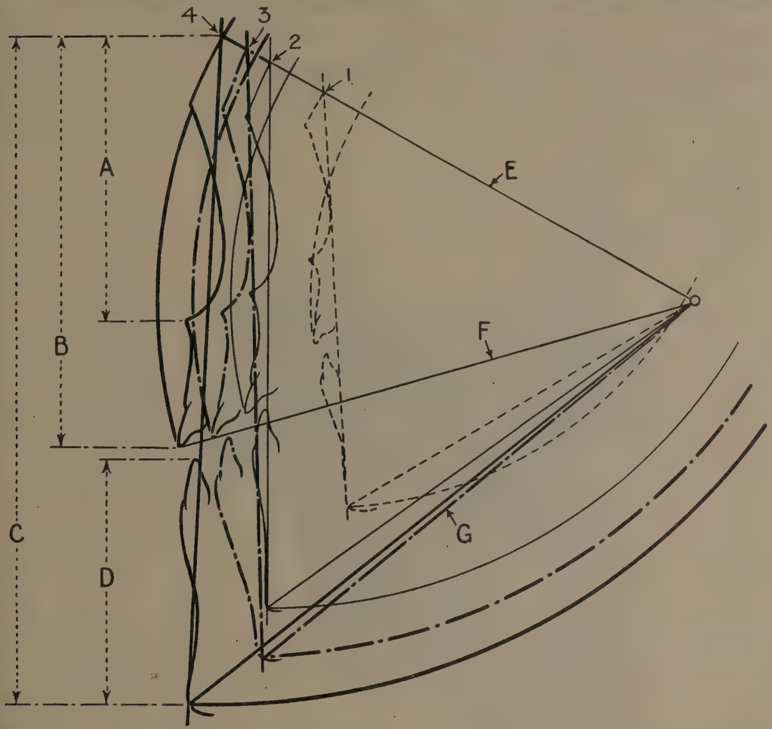

Fig. 88.—Diagram showing vertical and forward growth of the normal or sub-normal face. Vertical measurements taken from nasion; forward movements from trans-meatal axis. The arcs centred at the meatal axis show the progressive growth forwards along line F in relation to that along line E. The arcs centred at the nasion show the progressive vertical growth along line C in relation to that along line E. (Figs. 88 to 90 have been made from drawings kindly lent by A. Keith.)

Profile 1.—From child aged 3 years 6 months.
,, 2.—Represents mean of 10 children aged 9 to 11 years.
,, 3.—Represents mean of 9 children aged 15 to 16 years.
,, 4.—Represents mean of 10 adults.

of the maxilla—the area lying in front of the pterygo-maxillary fissure, where the molar part of the alveolar margin is produced—and the palate bone, new tissue being added on both anterior and posterior margins.

The deposit of bone in the suture between the maxillary and palate bones takes place, in the authors' opinion, at a greater rate than at the posterior part of the maxilla. Growth also takes place along the palatal or lower border of the vomer to keep pace with growth in front of the pterygo-maxillary fissure.

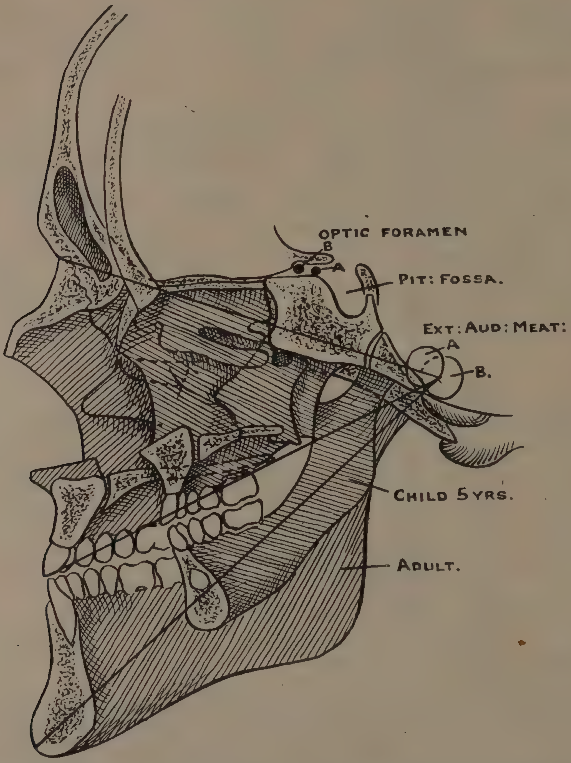

FIG. 89. (Figs. 88 to 90 have been made from drawings kindly lent by A. Keith.)

($\frac{2}{3}$ natural size.)

A.—Sagittal mesial section of the skull of a child with M_1 about to erupt, and estimated to be five years of age.

B.—Corresponding section of skull of adult Englishman. The skulls are superimposed so that the pituitary fossæ and cribriform plates correspond. The sections show the amount of growth in the sagittal mesial plane from the fifth to the twenty-fifth years.

Along the line G growth is chiefly in the region of the condyle and, in a small degree, in the region of the chin.

Growth also occurs at various superficial sites and sutures, at the junction of the temporal and malar bones, the addition being equal with that laid down on the posterior part of the maxilla.

At the malo-maxillary suture the growth is less. Important growth takes place at the fronto-nasal process, along the supra-orbital margin, and at the external angular process.

The authors' observations show that the great coronal system plays an important part in the growth of the jaws; this system includes the coronal suture itself, the temporo-malar, the fronto-sphenoid, spheno-ethmoid, pterygo-maxillary, and transverse suture of the palate.

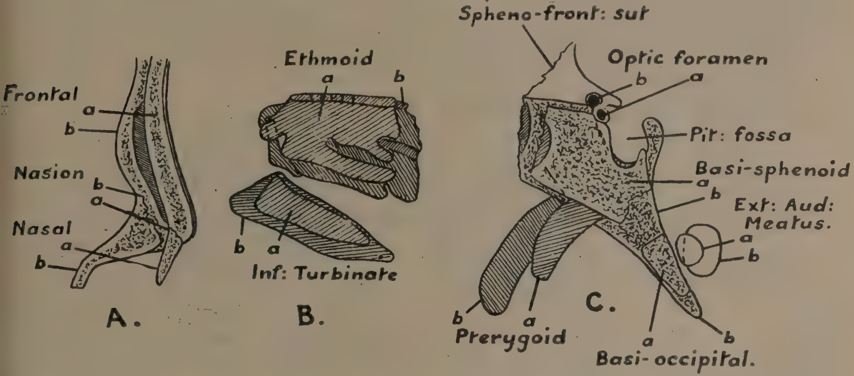

FIG. 90. (Figs. 88 to 90 have been made from drawings kindly lent by A. Keith.) ($\frac{2}{3}$ natural size.)

Sites of growth along line E in basal parts of skull: (*a*) child, aged 5 (see fig. 89); (*b*) adult (see fig. 89). A, fronto-nasal element; B, ethmoidal element; C, sphenoidal element.

By superimposing the skull of a prehistoric inhabitant of these islands with a well-developed palate upon the skull of a modern inhabitant with a contracted palate, the authors show that the contraction of the palate in the latter case is due to a general lack of growth of all the parts concerned in the general growth of the face.

The conclusion they reach is that the so-called adenoid face is due not to simple mechanical causes but to an arrest or disturbance of the elaborate machinery which underlies facial growth.

'*The normal orderly arrangement of the teeth in the jaws is the result of normal growth of the bone.* The teeth may react on the bone, but the main factor in the translation of the teeth to their normal position in the arch is the work of the growing bone. In other words, the arrangement of the teeth in a normal arch is not necessarily dependent on want of room in that arch.'[1] (Further reference will be made to this point in dealing with adenoids.)

(3) FACTORS INFLUENCING THE GROWTH OF THE JAWS.

The growth of the jaws is materially affected by functional activity. It will be convenient to deal with this subject under the following heads :—

(*a*) The influence of mastication.
(*b*) The function of the teeth.
(*c*) The action of the tongue.
(*d*) The nasal function.

It may here perhaps be pointed out that the proper function of a part is necessary for the due growth of that part, as instanced by the mal-development of the orbit which follows the loss of an eye in youth.

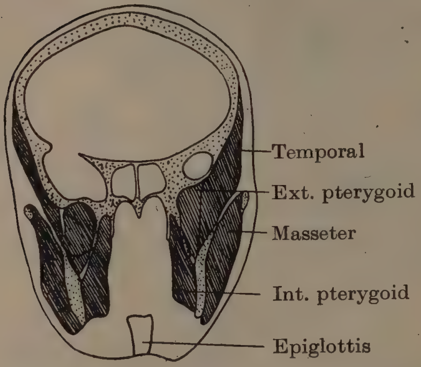

FIG. 91.

(Copied from the *Trans. Odonto. Soc.*, by kind permission of Harry Campbell.)

(*a*) **The Influence of Mastication.**—In considering the influence of mastication on the jaws a brief reference must be made to the relation of the muscles to the bones. Fig. 91 is a vertical transverse (slightly oblique) section through the head on a level with the epiglottis, showing the massiveness of the masticating muscles. Fig. 92 is a horizontal transverse section about an inch below the condyles of the mandible, showing the outward direction of the external pterygoids as well as the close relation of the levatores and tensores palati with the internal pterygoids. From these figures it will be seen that 'the pterygoids, when in a state of contraction, must exercise an outward and backward tug on the external pterygoid plate, palate bone, body of the sphenoid and maxilla, and tend to pull them away from the sagittal plane of the head.'

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[1] J. G. Turner, *Dental Cosmos*, xlvii, 43.

Harry Campbell is of the opinion that, in addition to the actual mechanical action of the pterygoid muscles, their rhythmic contraction greatly aids the circulation of the blood and lymph and so exercises a beneficial effect on the structures in the immediate neighbourhood.

(*b*) **The Function of the Teeth.**—The positions occupied by the teeth influence the growth of the jaws. The teeth transmit the force of muscular action to the bones. Normally, the maxillary teeth are arranged with the occluding surfaces of the premolars and molars facing slightly outwards, and the force applied to them in this position in mastication has a general tendency to expand the jaws. The more vertical the direction of the teeth the less will be the influence of muscular pressure towards increasing the width of the maxillæ.

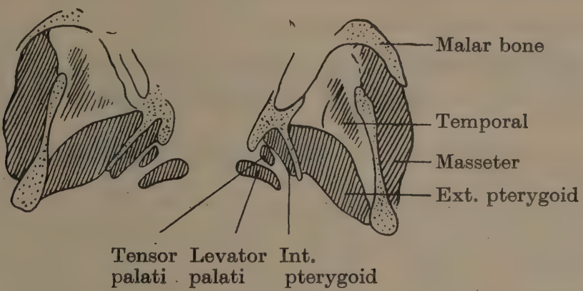

Fig. 92.

(Copied from the *Trans. Odonto. Soc.*, by kind permission of Harry Campbell.)

(*c*) **The Action of the Tongue.**—The tongue influences the position occupied by the teeth in the arch. Sim Wallace [1] considers that the tongue is the common denominator for the correlation of the upper and lower teeth, and that there is an intimate connection between the size and growth of the tongue and the size and development of the jaws. A large tongue, by causing the teeth to occupy a large arch, stimulates the outward growth of the jaws. The pressure of the tongue on the anterior part of the palate during the act of swallowing is probably an important factor in modifying this part of the mouth. In Wallace's opinion the under-development of the tongue and the decreased size of the jaws at the present day are to be attributed to the slight amount of mastication which is now necessary in the consumption of food.

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[1] An account of Sim Wallace's views is to be found in his work on 'Irregularities of the Teeth.'

(*d*) **The Nasal Function.**—The perfect performance of nasal respiration is an important factor in the growth of the maxilla. The use of the muscles of the nose influences the growth of the premaxilla, and the constant flow of air through the nasal passage stimulates the supply of blood to the mucous membrane lining the nasal fossa and the accessory sinuses and so stimulates the general growth of the bone.

(B) THE CLASSIFICATION OF ABNORMALITIES IN POSITION OF THE TEETH.

(1) ETIOLOGICAL CLASSIFICATION.

Abnormalities in position have been classified in several ways; they may be classified: (1) according to their etiological factors; (2) according to the type of occlusion which is present when they occur; this latter method is usually referred to as Angle's classification, from E. H. Angle, its originator. Both of these classifications are valuable, the former because every type of abnormality of position or occlusion is included, and because, as treatment rationally consists in the removal of the cause, it is a valuable guide to what treatment each case should receive. The latter is of value as conveying in a few words what the appearance of the patient may be in each case. It does not, however, cover all abnormalities in position or occlusion, nor does it give much etiological information or guide to the treatment of any given case. It deals only with the classification of occlusion as it is concerned with the antero-posterior position of the teeth, and in particular with the occlusion of the permanent first molars. It is founded on the uncertain factor of one tooth, the maxillary permanent first molar, being constant in medio-distal position.

Adami, in his excellent work on Pathology,[1] makes some valuable remarks in the introduction to the section on 'Causes of Disease,' and these remarks may with advantage be quoted here, because they will render more intelligible to the reader the account to be given of the causes producing irregularities in the position of the teeth. He states that 'Every departure from the normal, whether in the cell, the organ, or the system in general, is a pathological condition, provided that we recognise that the "normal" is not an absolutely fixed point, but is the expression for the limits between which the majority of the individuals of a given species will be found to group themselves as regards any particular attribute. Such pathological conditions must, it will be seen, be of two orders: either primarily due to some constitutional defect transmitted from the parent or parents (included with which we must place the effects of imperfections in the fusion of the male and female elements at the moment of fertilisation. Such effects, being associated with the actual constitution of the individual, are of internal origin and inherited); or, in the second place, they may be the result of some influence which first affects the individual after his genesis. Such conditions are of external origin and acquired. Morbid conditions, then, are to be classified into inherited and acquired.'

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[1] 'The Principles of Pathology,' by J. G. Adami.

It is advisable to make quite clear what is understood by the term 'inherited' as here used. To quote Adami, 'That alone is inherited which is the property of the individual at the moment of his becoming an individual, which is part and parcel of the paternal and maternal "germ plasm" from which he originates, or is provided by the interaction of the same.' 'It is unnecessary to point out, save as a precaution, that what is a property of the individual from the moment of beginning existence need not show itself for long years—a family failing towards premature baldness not until years after puberty, or an inheritance of gouty tendencies not until after thirty-five. As the different organs and parts assume their particular conformation and properties at different periods, and do not develop *pari passu*, so must the various inherited peculiarities make their appearance at various times.'

The classification based on these principles and adopted by Adami is as follows:—

(1) Inherited, due to influences affecting the ovum or the spermatozoon before or at the moment of fertilisation.

(2) Acquired.

(*a*) Ante-natal or of intra-uterine acquirement.

(*b*) Parturient, acquired at the time of birth before

Etiological Classification of Abnormalities in Position of the Teeth.

- (A) *Endogenous Causes* (All are congenital and some it is known may be hereditary.)
  - (*a*) Involving dental formative organs.
    - (α) Teeth in excess of the normal number.
    - (β) Absence of one or certain teeth.
    - (γ) Abnormal growth of the tooth band (misplaced teeth).
    - (δ) Abnormal development of teeth in size.
    - (ε) Abnormal development of teeth in form.
  - (*b*) Involving bone . . .
    - (α) Defective intra-uterine development (of one or both jaws).
    - (β) Variation in jaw development in relation to teeth of normal size.
    - (γ) Variation in degree of development of—
      - (i) Maxilla (and possibly associated bones).
      - (ii) Premaxilla.
      - (iii) Mandible.
  - (*c*) Involving soft parts. . Abnormal development of the fræna labiorum.
- (B) *Exogenous Causes.*
  - (*a*) Parturient
    - (α) Abnormal fœtal presentation } leading to excessive moulding.
    - (β) Abnormal maternal passages } leading to excessive moulding.
    - (γ) Application of forceps
  - (*a*) Trauma. . . . .
    - (i) Direct injury .
      - Partial dislocation of teeth.
      - Premature removal of teeth.
        - Deciduous.
        - Permanent.
      - Fractures of jaw.
      - Operations for cleft palate, etc.
    - (ii) Indirect injury (causing displacement usually during eruption).
      - Habits.
      - Results of cicatrices.
      - Undue retention of normal deciduous teeth.

- (*b*) Post-natal
  - (β) Infective processes in connection with the teeth.
    - (i) Loss of tissue from caries.
      - Undue retention of pulpless deciduous teeth.
    - (ii) Infective processes the sequelæ of caries.
      - Granulomata.
      - Cysts.
      - Inflammatory obstruction and injury.
  - (γ) Conditions leading to abnormal growth of the jaws.
    - (i) Errors in feeding and environment.
      - Rickets in association with adenoids.
    - (ii) Errors in manner of feeding.
      - Hand feeding.
    - (iii) Disease . . .
      - Congenital syphilis.
      - Acromegaly.
      - Oxycephaly.
      - Progeria.
      - Achondroplasia.
  - (δ) Neoplasms.
  - (ε) Physical causes of uncertain origin.
    - (i) Partly erupted teeth.
    - (ii) Completely unerupted teeth.

NOTES.

Endogenous Causes.—It is possible that those involving bone may be influenced by endocrine imbalance, maternal dietary deficiencies, or some other factor at present unknown.

Misplacement.—Primary misplacement of a tooth is a developmental error.

Displacement.—A displaced tooth necessarily once occupied another position, presumably its normal one. After a tooth has been displaced it is misplaced, but this is a secondary misplacement and quite different in origin from a primary misplacement.

The student must realise that some of these causes are very common and others very rare. Of the endogenous causes, the first three involving dental formative organs (α, β and γ) and the third involving bone (γ) are comparatively common; and of the exogenous post-natal causes, premature removal of teeth, habits, undue retention and early loss of deciduous teeth, rickets in association with adenoids, and hand feeding account for the majority of cases requiring orthodontic treatment.

complete separation of the individual from the maternal organism.

(c) Post-natal, acquired after birth.

There are objections to the application of this scheme to irregularities of the teeth. For example, it is often impossible to say with certainty whether a particular condition is due to an inborn predisposition or to an extrinsic stimulus; thus some cases of cleft palate are definitely hereditary, while others are without doubt traceable to defective nurture. From a biological point of view, however, the scheme has the great advantage of being based on **endogenous causes** (originating within the body) and **exogenous causes** (due to outside factors), and it is proposed to adapt this classification in describing the various abnormalities in the position of the teeth.

(A) ABNORMALITIES DUE TO ENDOGENOUS CAUSES (ALL ARE CONGENITAL AND SOME, IT IS KNOWN, MAY BE HEREDITARY).

(a) Involving Dental Formative Organs.

(α) Teeth in Excess of the Normal Number.

When supernumerary teeth are present in the arch, the normal arrangement of the teeth is necessarily disturbed (see fig. 93). Supernumerary teeth may erupt in the arch and cause perverted growth and non-eruption of the normal teeth. Thus, in fig. 94 the maxillary right incisor is placed high up in the alveolar process, with its root considerably curved.

(β) Absence of One or Certain Teeth.

In a similar way absence of one or more teeth also causes abnormality in the arrangement of teeth so that occlusion is not normal, though under the circumstances it may be good. In such a case as absence of all the third molars it is conceivable that it is possibly an advantage in the smaller jaws of civilised peoples; in such case the occlusion of the remaining teeth may be normal, in so far as it goes.

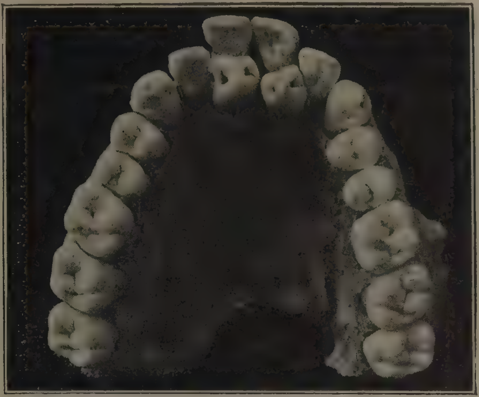

Fig. 93.—Maxillæ with two supernumerary teeth causing irregularity of the first incisors.

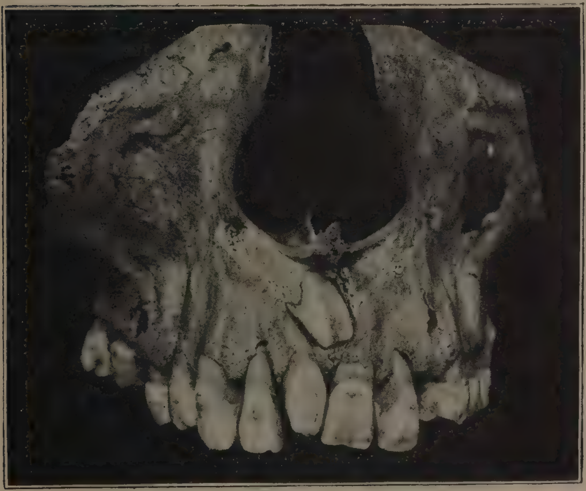

Fig. 94.—Maxillæ with the right first incisor embedded in the bone. A supernumerary tooth is present in the arch.

(γ) Abnormal Growth of the Tooth Band. (Misplaced Teeth.)

The irregularities included in this group seem to be due to the tooth bud being given off in an abnormal position.

Maxillary Canines.—Developmental irregularities in the position of this tooth are common, and vary from a slight deviation from the normal to complete inversion of the tooth and eruption into the nasal fossa. This irregularity was noted in seventeen out of 1,000 men examined. In one case both maxillary canines were misplaced, and in another case the maxillary left and the mandibular right canines were affected. The following specimens show a progressive degree of irregularity :—

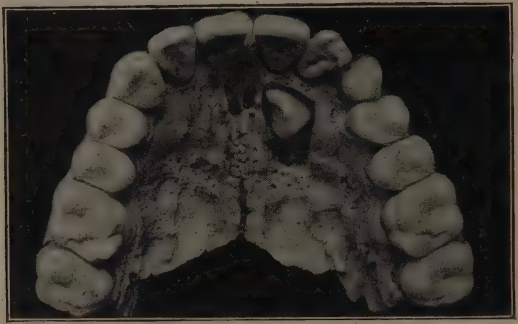

FIG. 95.—Maxillæ with the left canine erupting internal to the arch.

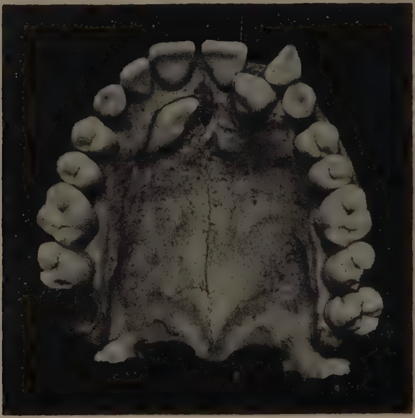

FIG. 96.—Maxillæ showing misplaced canines.

In fig. 95 the canine has erupted internal to the arch and caused slight irregularity in the position of the second incisor. In fig. 96 the left canine has erupted in a vertical position internal to the arch, whilst the right canine has assumed an oblique direction. A further stage is marked by the misplaced tooth lying external to the dental arch in a horizontal position, as seen in fig. 97. The tooth may take an upward turn (fig. 98), piercing the gum high up on the alveolar process ; and, finally, there may be complete inversion (fig. 99)—both teeth are here embedded in the nasal process of the maxillary bones. In the specimen shown in fig. 100 the tooth has erupted into the nasal fossa. In some of the foregoing examples the canine has failed to erupt. In other cases of extreme misplacement, however, the canine erupts but assumes a position in the arch which should normally be

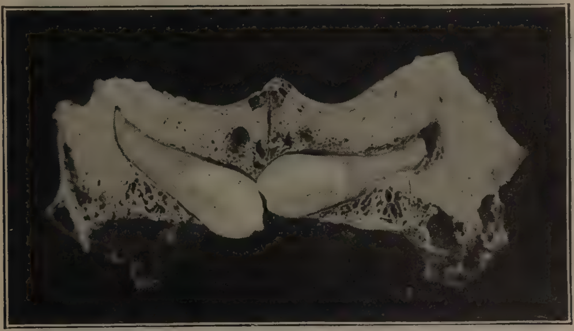

FIG. 97.—Portions of the maxillæ showing the canines lying in a horizontal direction with the crowns directed towards the median line.

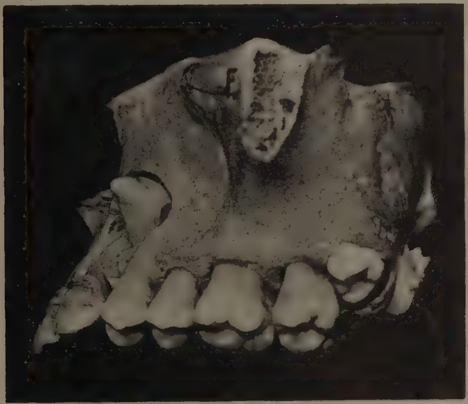

FIG. 98.—Left maxilla with the canine erupting in an upward direction.

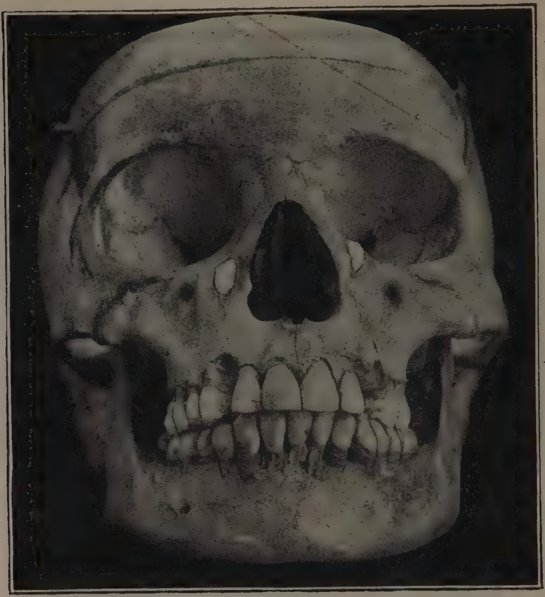

FIG. 99.—Skull showing the canines embedded in the nasal processes.

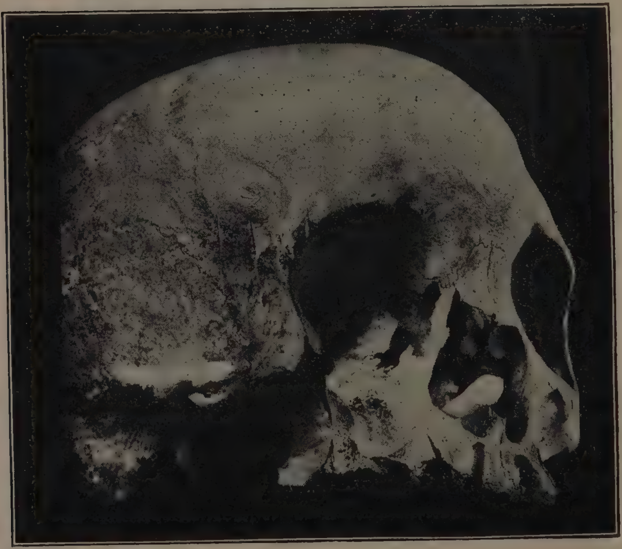

FIG. 100.—Skull of a negress with the right canine erupting in the nasal fossa. This specimen is in the Hunterian Collection.

occupied by another tooth. This irregularity is generally termed 'transposition.' In such cases the canine and second incisor usually change positions, and less commonly the canine and first premolar are transposed. In rare instances the canine may erupt posterior to both premolars. These abnormalities are at times hereditary.

Maxillary Premolars.—Complete misplacement of the premolar is comparatively a rare irregularity. An example is shown in fig. 101; sometimes this is associated with retention of the deciduous second molar and may then be a displacement.

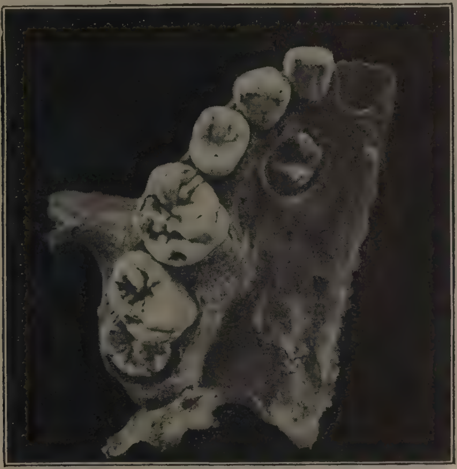

FIG. 101.—Right maxilla with the first premolar erupting in the palate.

The complete misplacement of a premolar is more common in monkeys than in man, and a study of the abnormality in these animals enables us to formulate a probable explanation of the cause. It will be noted that in monkeys the premolar, at times, develops well above the root of the deciduous molar (see fig. 102), and it can readily be understood, therefore, that if, in development, there is the slightest deviation from the normal direction, the tooth will erupt misplaced. The misplacement of a premolar in man is possibly to be attributed to similar conditions, that is to say, the premolar, instead of developing within the roots of its predecessor, is formed beyond them, and has therefore a tendency to assume an abnormal position in its subsequent growth.

Complete rotation of the second premolar occurs, the palatal aspect being placed outwards. This abnormality is sometimes symmetrical. In fig. 103 the second premolars have the external cusps rotated forwards.

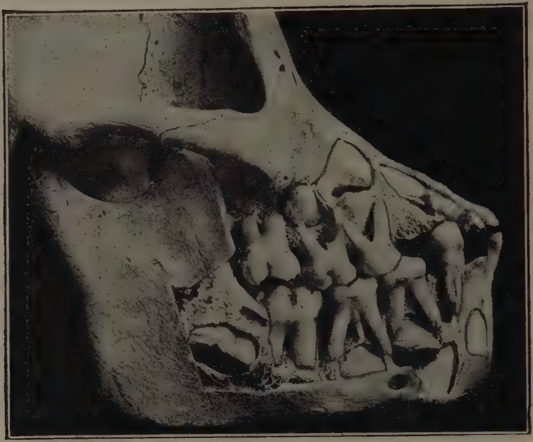

FIG. 102.—The skull of a chimpanzee to show the position of the developing second premolars.

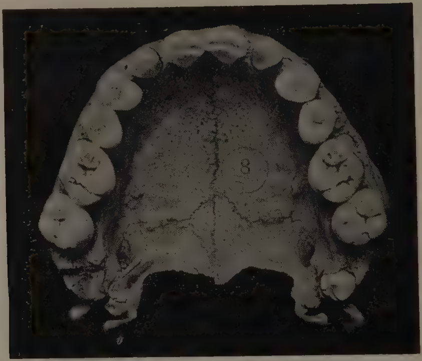

FIG. 103.—Maxillæ with the left third molar displaced backwards.

Maxillary Molars.—The maxillary third molar is frequently misplaced. This tooth may be carried away from the second molar (see fig. 103), but more often it is situated high in the tuberosity and, owing to insufficient bony growth to allow of its eruption, becomes embraced by the roots of the second molar. The crowns may be tilted forwards and the roots backwards, or the tooth may be rotated, the buccal surface facing forwards. Lastly, we get that singular condition in which the crown of the third molar lies in an inverted position within the roots of the second molar (fig. 104).

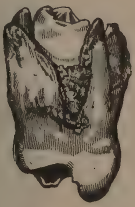

Fig. 104. (From 'A System of Dental Surgery,' C. S. Tomes and W. S. Nowell.)

A remarkable specimen showing irregularity in position of several teeth is shown in fig. 105. The maxillæ and the rest of the skull are normal in character, but the teeth show an extraordinary arrangement. On one side the canine has erupted between the premolars, with the root of the deciduous canine persistent in its normal position. On the other side the canine lies obliquely across the alveolar ridge, the crown pointing forwards and slightly outwards and downwards; the tooth is rotated so that the internal aspect of the crown is downwards. The premolars are missing, and there are no signs that they were ever present. The first molar lies horizontally, with the occlusal surface directly backwards and the roots forwards. It is of interest to note that there are two enamel nodules on the left second molar.

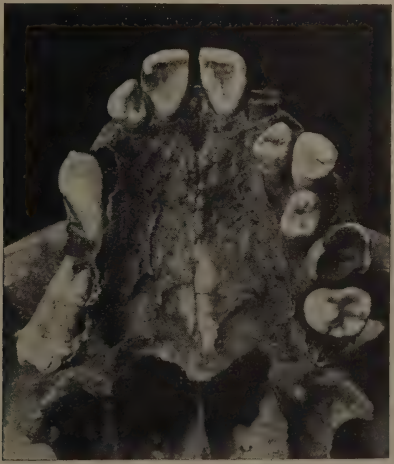

Fig. 105.—Portion of a skull showing multiple irregularities in position of the teeth.

Mandibular Incisors.—In well-developed arches the second incisor is at times rotated so that the distal angle looks inwards. This abnormality is sometimes inherited. A mandible showing this abnormality is illustrated in fig. 106, and it is interesting to note that the developing second molar is also misplaced in this specimen.

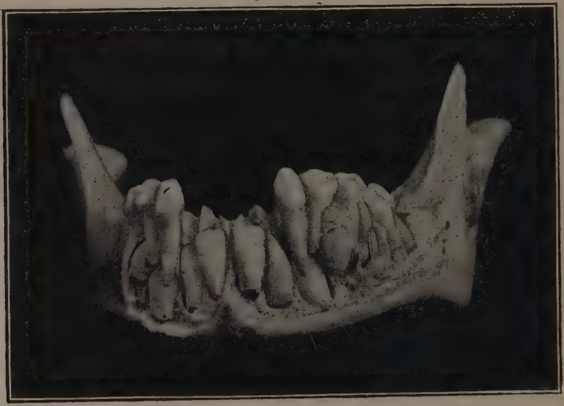

FIG. 106.—Mandible with the left second incisor rotated.

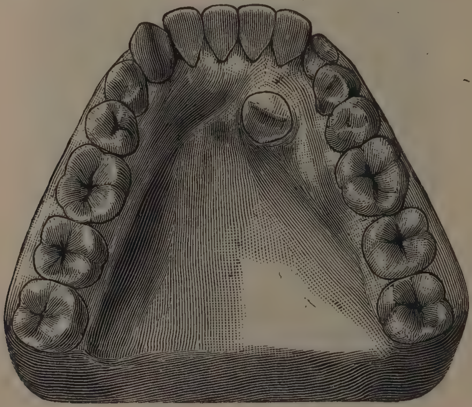

FIG. 107.—Model showing the mandibular right canine placed internal to the arch.

Mandibular Canines.—Although not so prone to be developmentally misplaced as its fellow maxillary tooth, the mandibular canine shows definite variations in its position. It may erupt internal to the arch (fig. 107), or it may appear external to the arch directly in front of the incisors (fig. 108), while in other instances it may remain embedded in the bone, as in fig. 109. In one case observed, the right canine erupted between the first and second incisors. In 1,000 cases examined, fourteen examples of misplaced unerupted mandibular canines were noted ; in one instance the condition was associated with a misplaced maxillary left canine.

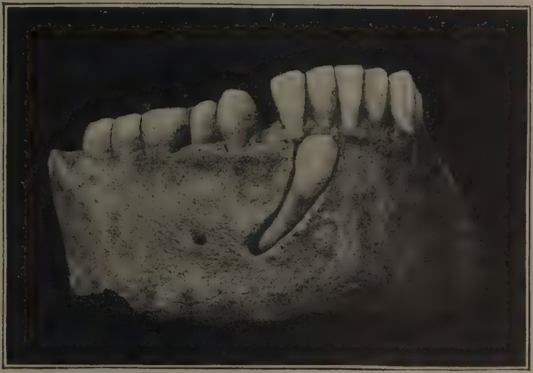

FIG. 108.—Mandible with the right canine erupting anterior to the roots of the incisors.

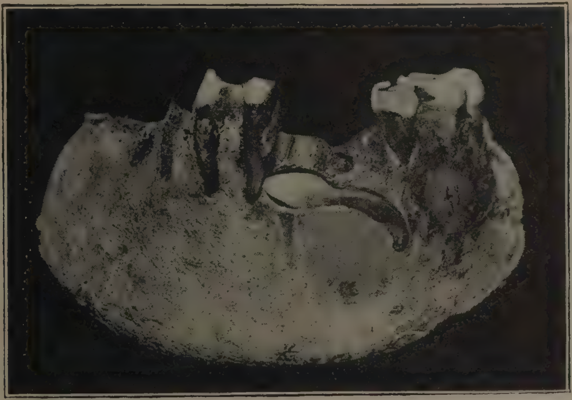

FIG. 109.—Portion of mandible with the left canine embedded in the bone.

Mandibular Premolars.—These teeth are at times completely misplaced. In the specimen shown in fig. 110 the first premolar lies in a horizontal position embedded in the bone with the crown pointing away from the mid-line. Both the second premolars are occasionally found to be embedded in the bone (fig. 111). It will be seen from the illustration that the roots of the teeth are in close proximity to the lower border of the bone. There is ample room in the arch, and the condition cannot therefore be a displacement due to impaction.

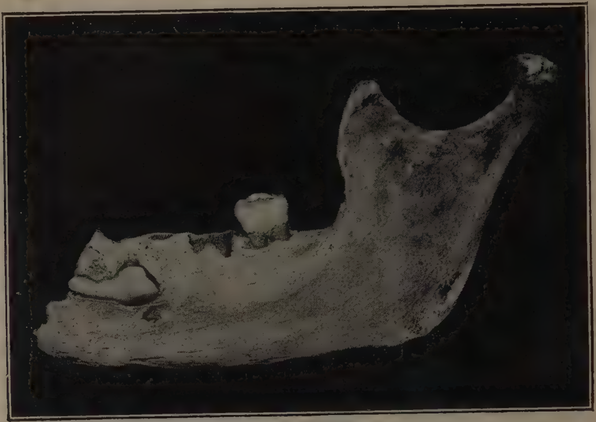

FIG. 110.—Left half of mandible with the first premolar embedded in the bone.

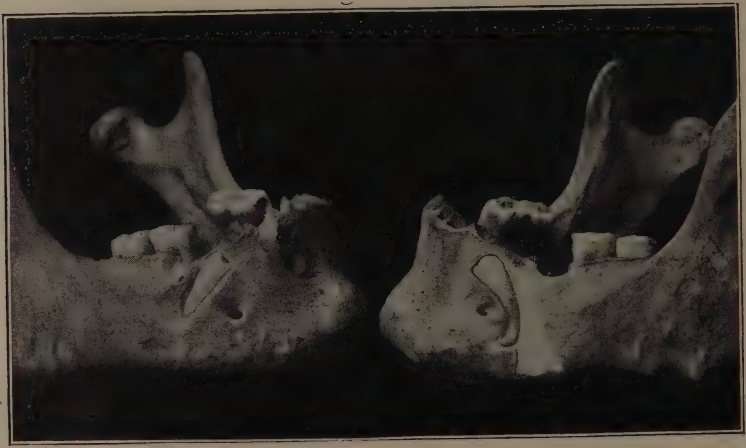

FIG. 111.—Mandible with the second premolars embedded in the bone.

Mandibular Molars.—A curious inward tilt of the second molar, which is sometimes met with, should, it seems, be regarded as an inherited or congenital variation. In some instances the tooth occludes completely internal to the upper arch. The second molar normally develops with an inward tilt, but occasionally the tilt is more pronounced and the development takes place with the occluding surfaces directly inwards and completely internal to the upper arch. The condition is frequently seen in anthropoidea (fig. 112). The diagram (fig. 113), taken from a radiogram, shows the second and third molars misplaced. The second molar is tilted, the crown looking forwards, while the third molar has the occlusal surface looking downwards.

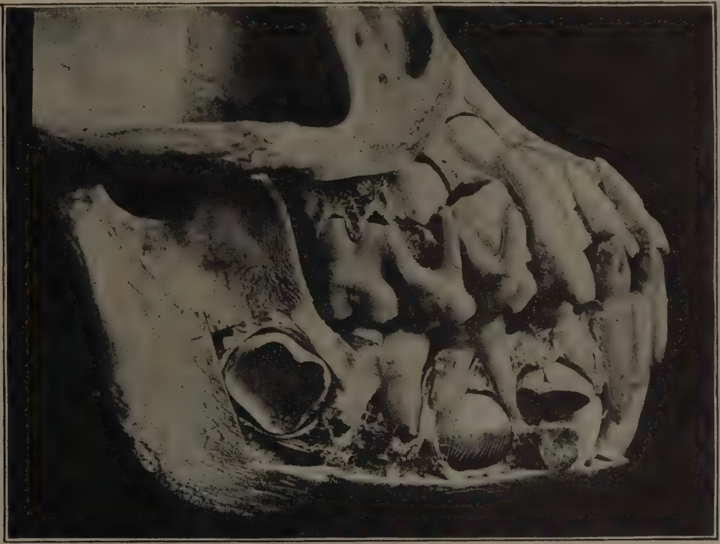

Fig. 112.—Skull and mandible of a young ape, to show the position of the developing permanent second molars.

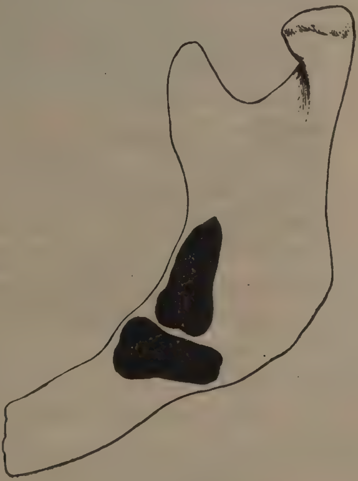

Fig. 113.

There are few conditions requiring treatment in practice which demand more thought and care than a misplaced mandibular third molar that is causing trouble. The tooth may be only slightly tilted forward (fig. 114), or it may be placed horizontally, the occluding surface of the crown impinging on the posterior root of the second molar (fig. 115). In some instances the tooth may be so placed that the occluding surface faces downwards and inwards. An examination of specimens exhibiting the mandibular third molar misplaced will demonstrate very clearly that the roots are to a greater or less extent covered by the ramus of the mandible. This is well shown in fig. 113. Other points brought out in this specimen are the close proximity of the roots of the second and third molars to the mandibular canal, and the small amount of bone available at the posterior aspect of the second molar.

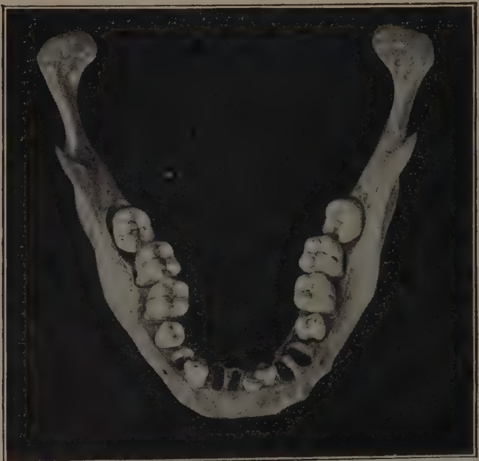

FIG. 114.—Mandible showing slight tilting of the third molars.

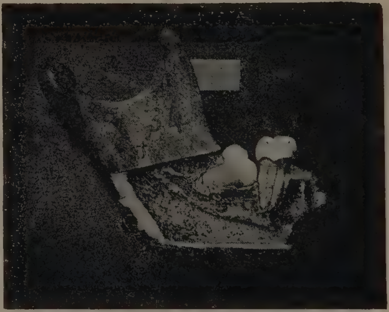

FIG. 115.—Portion of mandible with the third molar impinging on the root of the second molar. (From *Trans. Odonto. Soc.*)

By far the most interesting specimen of misplaced third molars is that shown in fig. 116. The right third molar is situated in the centre of the upper part of the ramus. The tooth is placed vertically, the crown being but little below the sigmoid notch. The left third molar is also misplaced, and rests in the anterior portion of the ramus. Both teeth lie above the mandibular canal.

J. J. Andrew [1] has recorded a case in which in a woman aged about fifty-five the mandibular left third molar was misplaced high up in the ramus, but with the apex towards the sigmoid notch.

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[1] *Brit. Dent. Journ.*, xxx, 289.

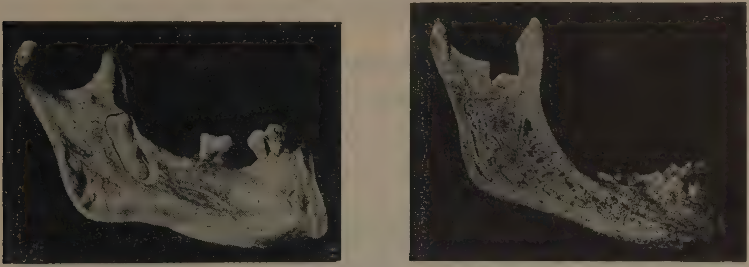

FIG. 116.—Mandible with the third molars situated in the rami. (From *Trans. Odonto. Soc.*)

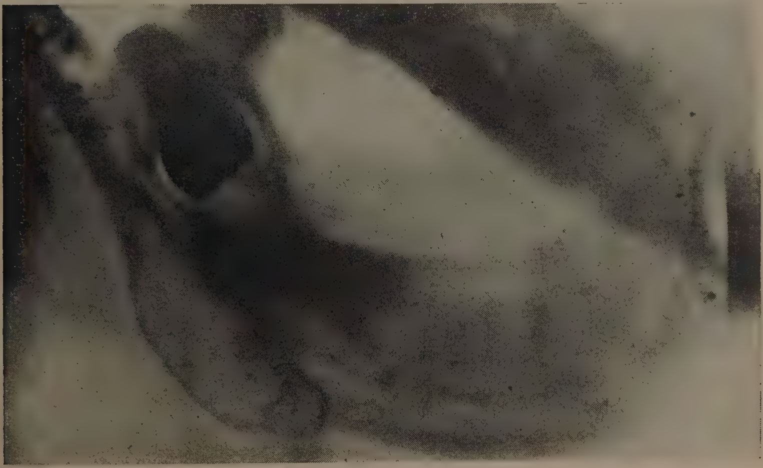

FIG. 116A.—Mandible with third molar inverted in ramus. (H. Stobie.)

In the case shown in fig. 117 the third molar erupted at the angle of the jaw.

Misplaced mandibular third molars are met with in monkeys and apes. It is a fairly common irregularity in man, thirty examples being seen in one thousand patients, the left tooth being misplaced in twenty-four cases and the right tooth in ten.

(δ) Abnormal Development of the Teeth in Size.

Variation in the size of teeth may occasion abnormalities in position, whether the teeth are larger or smaller than normal. If they are *larger than normal throughout the dentition* and the arch is not correspondingly larger crowding must take place, and the displacements which take place will, other things being normal, largely be governed by the order in which the teeth erupt. Such cases are not uncommon.

Similarly, if *certain teeth only are larger*, the space they occupy may obviously throw neighbouring teeth out of their normal occlusion, and the same remark may apply, but not so necessarily, if individual teeth are smaller than normal.

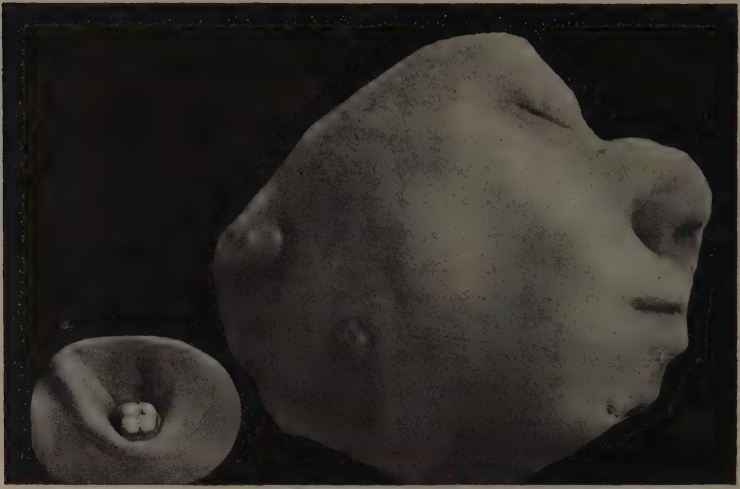

Fig. 117.

If the dentition as a whole is smaller than normal no abnormalities in position usually result.

A rare type of case of enlargement of certain teeth is recorded by Martin Rushton[1] as ‘ Partial gigantism of face and teeth ’ in a boy aged 5½ years, in whom many of the teeth, both deciduous and permanent, were larger on one side than the other, so that the midline was deflected to the unaffected side (fig. 118). This case is, of course, not purely one affecting the teeth only, as bones and soft parts on the affected side were also enlarged, including the ear, palate, alveolar processes, lips, nostril, cheek and part of the tongue. Rushton gives details of three other similar cases : in all of them the abnormality was noticed at birth, and therefore starts *in utero*; there is no evidence that it is familial. (See also p. 19.)

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[1] *Brit. Dent. Journ.*, 1937, lxii, 572.

(ε) Abnormal Development of Teeth in Form.

It will be obvious that variations of this type, whether affecting one, many, or all teeth, may occasion abnormalities in position and occlusion; it is, however, not a common cause.

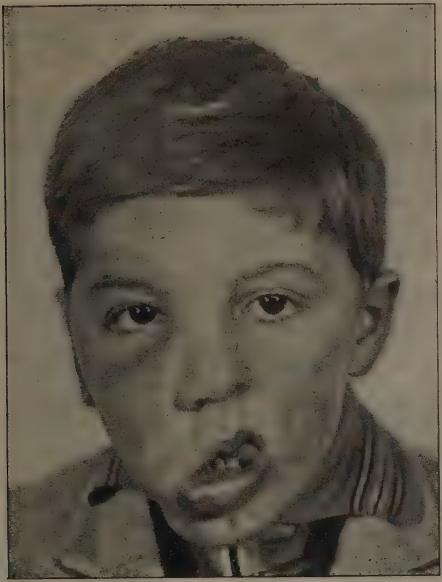

FIG. 118.—The two teeth visible are both right incisors. (M. A. Rushton.) (From *Brit. Dent. Journ.*)

(*b*) INVOLVING BONE.

(*a*) Defective [1] Intra-uterine Development.

In cases of defective development of the maxilla or mandible the teeth present marked abnormalities of position. In cleft palate involving the premaxilla the incisors and canines are misplaced, while partial arrest in the growth of the mandible naturally results in considerable disturbance of the relation of the dental arches to one another. A case of non-development of one mandibular ascending ramus is reported by Martin Rushton in

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[1] Defective: something wanting.

Brit. Dent. Journ., lxiv, 549. The abnormality was associated with other local developmental errors; other comparable cases have also been recorded.

(β) Abnormalities due to Variation of Jaw Development in Relation to Teeth of Normal Size.

'Individual variability is a general character of all common and widespread species of animals or plants; and, further, this variability extends, so far as we know, to every part and organ, whether external or internal, as well as to every mental faculty. Yet more important is the fact that each part or organ varies to a considerable extent independently of other parts' (Wallace).

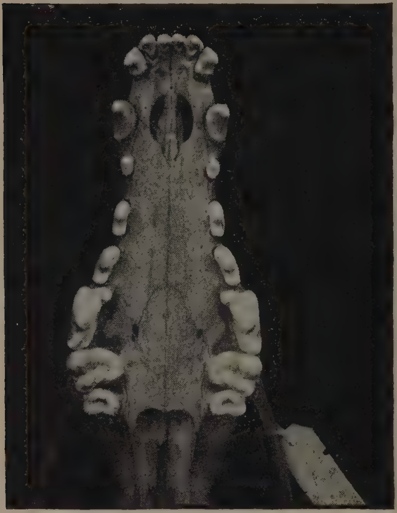

FIG. 119.—Palatal view of a Borzoi hound. (From the *Dental Record.*)

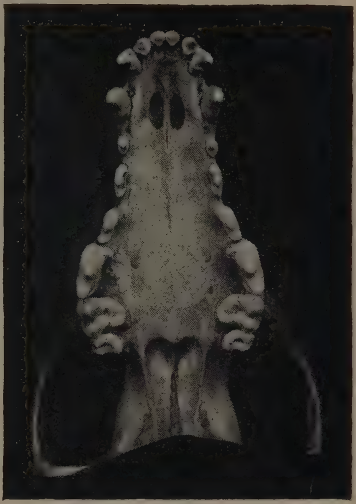

FIG. 120.—Palatal view of a greyhound. (From the *Dental Record.*)

In this section it is proposed to consider how far this phenomenon of variability is responsible for general irregularity of the teeth. If series of different species of animals be examined it will readily be observed that a diminution in the length of the jaws is not accompanied by a corresponding diminution in the size of the teeth, with the result that in many species the teeth are arranged in a slightly irregular or crowded manner. In animals under domestication this variation in the length of the jaws without corresponding adjustment in the size of the teeth is still more marked, particularly in the different breeds of dogs. It may be said that as a rule the shorter the jaw, the greater the tendency to crowding of the teeth.

As an illustration, four breeds of dogs will be compared, namely, the Borzoi hound, the Greyhound, the Aberdeen terrier, and the Bulldog. The length of the jaws of these animals decreases in the order shown. The maxillæ of the Borzoi hound (fig. 119) are very long and the anterior cheek teeth are well separated; in the Greyhound (fig. 120) the maxillæ are shorter, and there are spaces between the anterior cheek teeth, but these teeth are not so widely separated as in the Borzoi hound; in the Aberdeen terrier (fig. 121), with its still shorter maxillæ, the premolar teeth are in contact, and there is insufficient room for the fourth premolar; while in the Bulldog (fig. 122), with its comparatively short maxillæ, the premolars and molars are arranged in an extremely crowded manner.

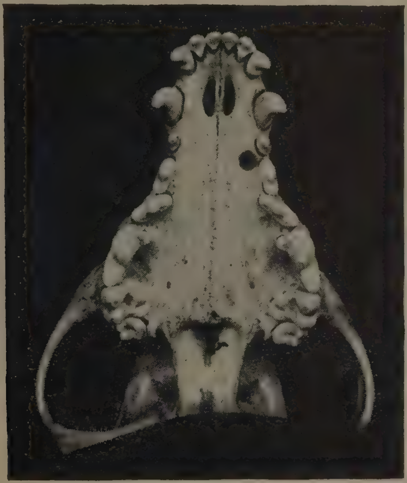

FIG. 121.—Palatal view of an Aberdeen terrier.
(From the *Dental Record.*)

Man is no exception to this law of variation, and with the progress of civilisation the reduction in the size of the jaw out of proportion to the reduction in the size of the teeth has been marked.

Talbot,[1] who has collected a mass of statistics relating to irregularities, affirms that the early Britons possessed maxillæ varying in their lateral diameters from 2·12 to 2·62 in., whilst in modern Englishmen the maxillæ vary from 1·88 to 2·44 in., the minimum diameter having thus decreased more than the maximum; and that a comparison of the maxillæ of ancient Romans with modern inhabitants of Southern Italy gives a similar result. From these data he infers that the size of the jaws has diminished in the inhabitants of England and Italy, but it is not quite clear that these comparisons have any etiological significance, seeing that modern Englishmen are only to a very small degree, if at all, descendants of the ancient Britons; and similarly, the present inhabitants of Southern Italy have but little claim to direct descent from the ancient Romans.

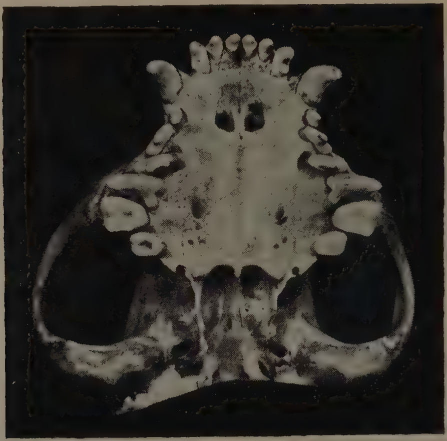

FIG. 122.—Palatal view of a bulldog.
(From the *Dental Record.*)

There is, however, a general consensus of opinion in favour of the view that the jaws of modern races are decreasing in size, and the teeth also, though in a lesser degree. It is interesting to note that it has been possible to trace a diminution in the size of the teeth in prehistoric times. W. Wright [2] has found that in the Neolithic and Bronze Ages the average length of the molar was greater than it was later on in the early Iron Age.

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[1] 'The Etiology of Osseous Deformities of the Head, Face, Jaws, and Teeth,' Third Edition, 67.

[2] *Brit. Dent. Journ.*, February 16, 1903.

The cause of the progressive diminution in the size of the jaws has not yet been clearly demonstrated, but, in view of all the ascertained facts bearing on the subject, the change would appear to be attributable in a great measure to the influence of civilisation, and more particularly to diet and to the manner in which food is prepared. Sim Wallace, who has closely studied the subject, considers that the diminution is not due to heredity, but is 'a characteristic developed in each generation as the result of the action of the environment.' Under present-day conditions the physical character of the foodstuffs is responsible for insufficient mastication, and thus by the 'action of environment' the jaws are deprived of the full stimulus which is essential for their complete development.

In the case of the animals referred to, artificial selection (breeding for points) has had a lot to do with the extremes of variation produced, but as regards man, environment seems to be the prime factor, and it has not yet produced such extreme degrees as in animals.

(γ) Variation in Degree of Development of: i. Maxilla (and possibly associated bones). ii. Premaxilla. iii. Mandible.

These variations produce cases of lack of occlusion of many teeth and involve variations in the :

i. Maxilla (and possibly associated bones), producing posterior open bite, and some cases of superior protrusion and posterior lack of occlusion.

ii. Premaxilla, producing anterior open bite and some cases of superior protrusion.

iii. Mandible, producing inferior protrusion, cross bite, and some cases of superior protrusion (or inferior retrusion).

Though this is a convenient method of classification, it will be found that somewhat similar abnormalities may be produced from different sources and, in some cases, by Endogenous and Exogenous causes acting together ; examples of this are found in the cases grouped as 'superior protrusion,' where the abnormalities look somewhat similar but have different, mixed, or uncertain causes ; the defect, too, does not always arise in the same place, but appears to arise sometimes in the maxilla, sometimes in the premaxilla, and sometimes in the mandible.

A curious but well-defined class of irregularity is shown in fig. 123. The maxillary premolars and the first and second molars on the right side have erupted external to the arch of the mandibular teeth. This irregularity did not exist in the deciduous dentition, and the first sign of it was the eruption of the maxillary right first molar in the position shown in fig. 124. Rarely such a

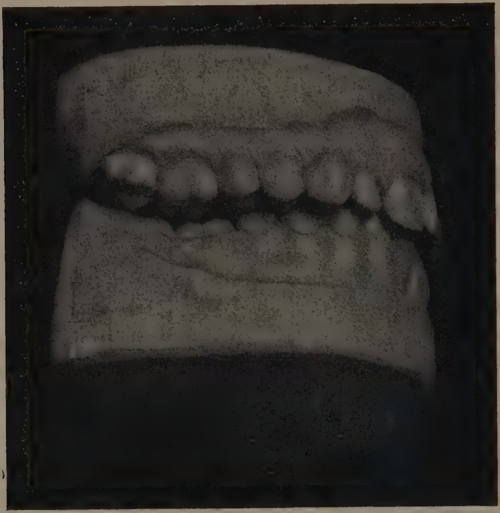

FIG. 123.—Models of a female at fourteen years, showing the maxillary premolars and molars external to the arch of the mandibular teeth. Posterior lack of occlusion.

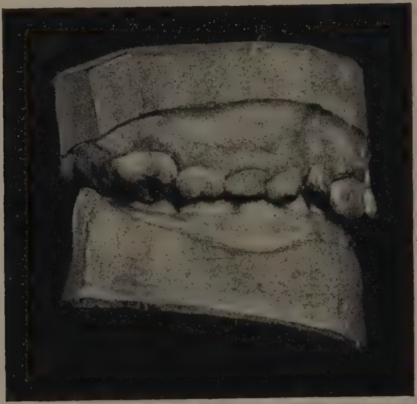

FIG. 124.—Models of the same individual taken at about seven years of age to show the occlusion of the deciduous teeth.

condition occurs bilaterally. A similar condition is occasionally seen in apes and monkeys.

The explanation of these cases is probably that of abnormal maxillary growth, though possibly it is an aberration of that portion of the tooth band from which the teeth have their origin.

To facilitate considerations of treatment this group of cases will be considered together and from the aspect of the deformity produced rather than the etiology (see Chapter VIII).

(*c*) INVOLVING SOFT PARTS.

Abnormal Development of the Fræna Labiorum.

Under this heading is included the irregularity of the first incisors where the *frœnum of the lip*, instead of blending with the mucoperiosteum on the anterior aspect of the alveolar process, is continued between the incisors and blends with the tissues in the region of the anterior palatine foramen. In these circumstances the frænum presses on the teeth with every movement of the lip, and the pressure tends to separate the teeth. A persistent labial frænum of the lower lip occurs, though much less frequently, and is at times hereditary.

(B) ABNORMALITIES DUE TO EXOGENOUS CAUSES (OUTSIDE FACTORS).

(*a*) PARTURIENT, ACQUIRED AT THE TIME OF BIRTH BEFORE COMPLETE SEPARATION OF THE INDIVIDUAL FROM THE MATERNAL ORGANISM.

These are grouped etiologically as due to :—

(α) Abnormal fœtal presentation } leading to excessive
(β) Abnormal maternal passages } moulding.
(γ) Application of forceps.

There is ground for believing that a few abnormalities of the teeth may be traced to injury at birth.

(α) Abnormal Fœtal Presentation.

The models shown in fig. 125 are those of a boy about five years of age. The parents are healthy, and possess normal jaws.

The child was breast-fed, and there was no suspicion of adenoids. The width of the arch is 22·76 mm. across the inter-canine region and 29·65 mm. across the second molar region. These dimensions are above the average. The occlusion is abnormal, the mandibular teeth being posterior to their normal relation to the maxillary teeth. The cause of this mal-occlusion was difficult to account for until it was ascertained that the child was an example of face presentation and that the labour had been prolonged. Before complete extension occurs in face presentation the head passes through the brow position and the diameter of engagement is the mento-vertical, *i.e.* from the point of the chin to the most distant point of the vertex (see fig. 126). Whilst this position is maintained the jaw must be compressed. As fuller extension occurs the occiput is compressed against the spine and the chin slips up, the diameter of engagement then being the cervico-vertical and cervico-bregmatic ; at this stage there is no undue pressure on the chin. It seems quite possible, therefore, that if labour is prolonged during the mento-vertical stage, permanent injury may be done to the mandible.

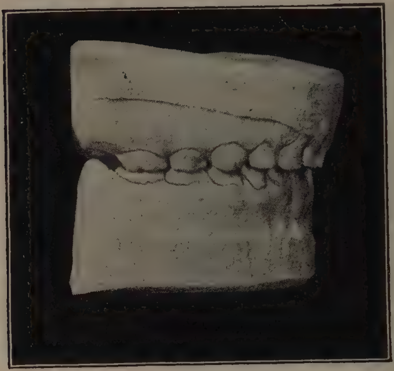
FIG. 125.—Models showing abnormal occlusion of the teeth.

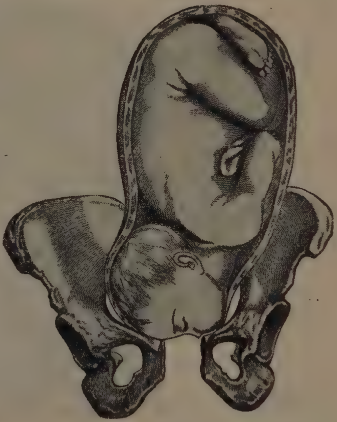
FIG. 126.—Diagram of a face presentation.

Figs. 127 and 128 are reproduced from Edgar's 'Practice of Obstetrics' and show the face before and after moulding. The appearance of the face after moulding suggests some retraction of the mandible.

Another of these cases is shown in fig. 129. The anterior maxillary teeth are unduly prominent. The child was breast-fed and there was no trace of adenoids. The occlusion is abnormal, the mandibular teeth being postnormal in relation to the maxillary teeth. In this case the labour was difficult and prolonged. The jaws of both parents are well developed, and the remaining

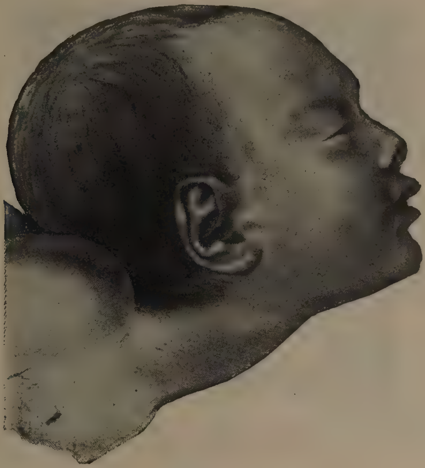

Fig. 127.
Shows the head before moulding.
(From Edgar's 'Practice of Obstetrics.')

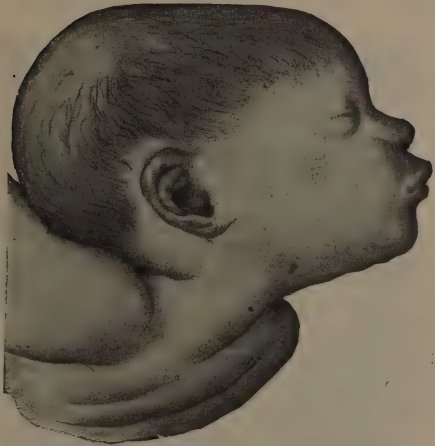

Fig. 128.—The head after moulding in a case of face presentation.
(From Edgar's 'Practice of Obstetrics.')

children in the family—four in number—have well-developed arches and normal occlusions.

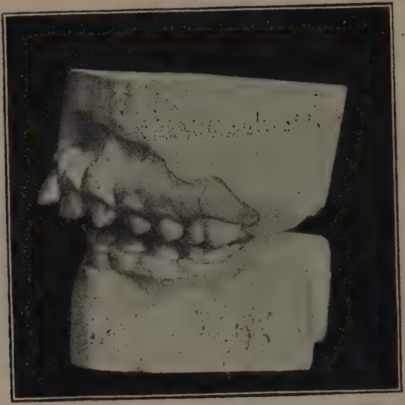

Fig. 129.—Models of a girl at eleven years with abnormal occlusion of the teeth associated with a difficult face presentation.

(β) Abnormal Maternal Passages.

Similarly, if the maternal passages are abnormally small or in any other way out of proportion or the child abnormally large,

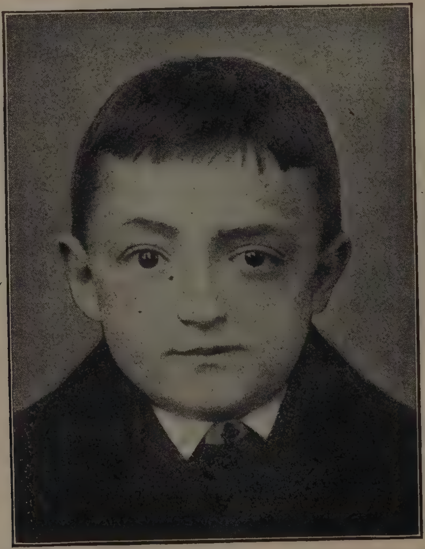

Fig. 130.—Photograph of a child with extreme asymmetry of the face.

excessive moulding may take place; sometimes, of course, these cases need the application of forceps in addition, and so may be further complicated.

Asymmetry of the dental arch is often seen and may occasionally be the effect of excessive moulding of the skull during birth. An example is shown in fig. 130. It will be noticed that the left side of the face is flattened and that the left occipital region is squeezed outwards. In this case the labour was prolonged, but no instrument was used. According to the father's statement, 'the child was born with the head as it is, but the doctor said it would come straight in time.' The asymmetry of the mandibular arch is shown in a drawing on mm. paper from a model of the mouth (fig. 131).

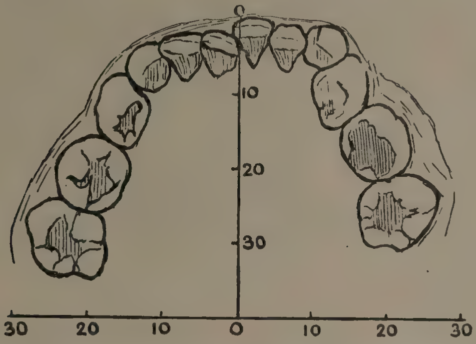

FIG. 131.—The mandibular teeth of the case shown in fig. 130.

(γ) Application of Forceps.

Fig. 132 illustrates the case of a boy six years of age who came under the care of G. Berwick. The defective growth of the mandible, due to an injury at birth, resulted in marked prominence of the upper teeth. The labour was very difficult and it was necessary to use forceps, which injured the face on the right side from the angle of the jaws to the chin.

Two cases of injury to one condyle of the mandible at birth, the left condyle in each case, with subsequent lack of growth and fibrous anchylosis on that side, were shown by E. A. Hardy at a clinical meeting of the Odontological Section of the Royal Society of Medicine on January 28, 1935: both had been breech presentations. The deformity in each case was apparently similar to those shown in figs. 133 and 134.

It seems highly probable that prolonged labour, especially in certain presentations, may lead to abnormality in position of the teeth.

The facts quoted above are sufficiently suggestive to warrant a full inquiry into a possible relationship between difficult labour and irregularities of the teeth.

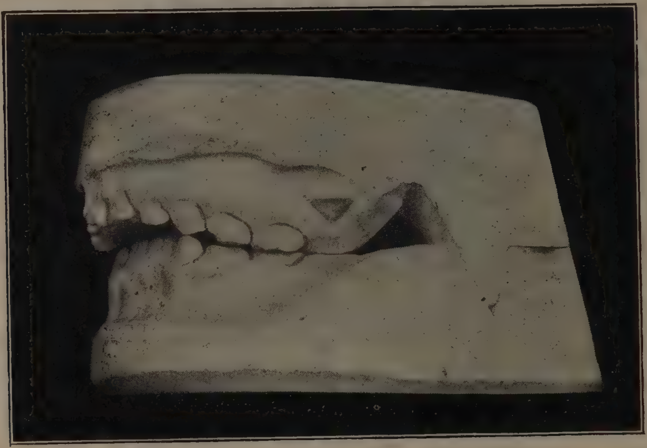

Fig. 132.—Models of a child, showing the results of injury of the mandible at birth.

(b) Post-natal or Acquired after Birth.

The majority of irregularities fall under this heading, and can be traced chiefly to (α) trauma, (β) infective processes in connection with the teeth, (γ) conditions leading to abnormal growth of the jaws, and rarely to (δ) neoplasms, and (ϵ) physical causes of unknown origin.

(α) Traumatic Causes.

The traumatic causes leading to deformities of the dental arch can be grouped under two headings :—

(i) Direct injury of the teeth or jaws.

(ii) Indirect injury (causing displacement usually during eruption).

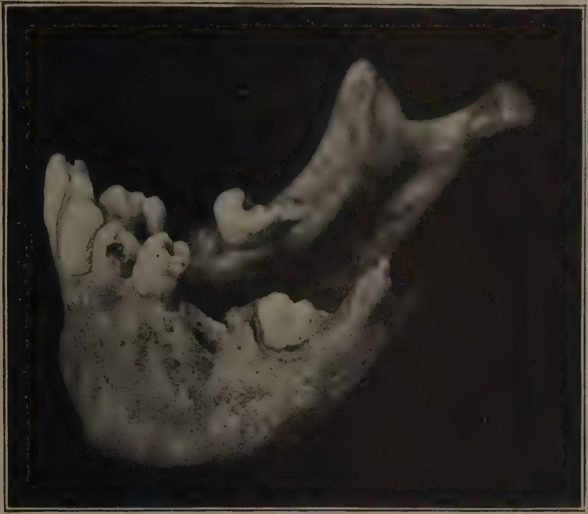

FIG. 133.—Specimen showing arrested growth of the left half of the mandible.

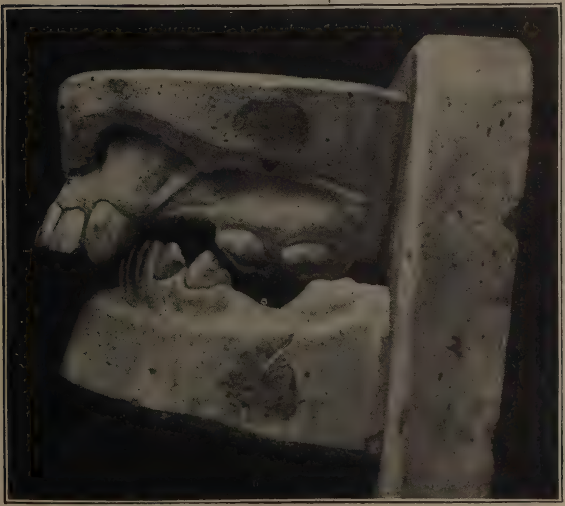

FIG. 134.—Models of a patient in which the left half of the mandible was arrested in its growth.

(i) *Direct Injury of the Teeth or Jaws.*

Incomplete Dislocation of Teeth.—The incomplete dislocation of a tooth or teeth if not corrected may result in permanent faulty position. For the possibilities which may arise from this condition and their treatment the student is referred to p. 290 *et seq.*

The Premature Removal of Teeth.—With the removal of a tooth there is a tendency for the approximal teeth to come together. In the maxilla, this movement is brought about by the muscular pressure of the lips, the tension of the cheeks and the forward movement of the permanent molars; in the mandible, by the action of the tongue and the tilting movement of the permanent molars. The extent of the deformity depends on the tooth removed, the time of removal, the occlusion of the teeth, and the development of the bones.

The following examples will illustrate these points. If a maxillary deciduous second molar is removed after the eruption of the permanent first molar, the latter tooth will tend to move or tilt (or both) forwards, and the deciduous first molar backwards. The extent of the movement of the permanent molar will vary according to the degree of interference with the growth of the bone in the molar region. If there has been normal growth, and if the first molar is in good occlusion with the mandibular tooth (*i.e.* interdigitation of the cusps is well marked), the amount of forward movement will be slight. If, on the other hand, there has been interference with the growth of bone, the tendency of the permanent molar to forward movement will be marked, even though the occlusion may be good. The tendency of the deciduous first molar to move backwards is brought about by the muscular pressure of the lips, but the lips will not exert much influence if the occlusion with the mandibular teeth is good.

If a deciduous second molar is removed before the permanent molar has erupted, the forward movement of the latter will be marked, and in some cases it will take up the whole space previously occupied by the deciduous tooth.

If a deciduous canine is removed at an early age, the closing of the space is mainly due to a spreading movement of the incisors, brought about in the maxillary arch by the lips and in the mandibular arch by both lips and tongue. The deciduous molar may also move forwards, but this will depend mainly upon the forward pressure of the permanent molars.

If the mandibular permanent first molars have been removed before the eruption of the second premolars, the latter occasionally erupt far back in the arch, as shown in fig. 135.

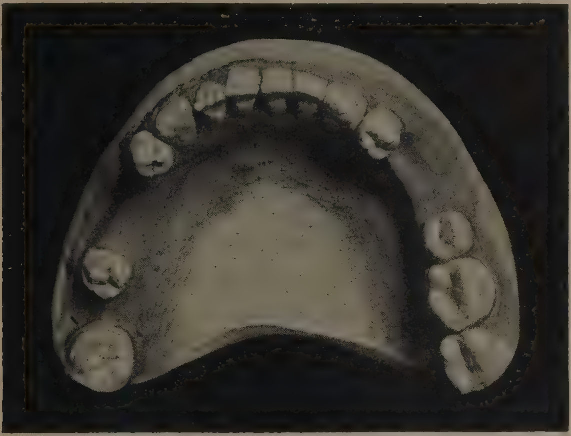

FIG. 135.—Model showing movement backwards of the mandibular second premolars following early extraction of the permanent first molars.

Fracture of the Jaw in children is frequently followed by irregularity in the position of the permanent teeth located in the region of the fracture.

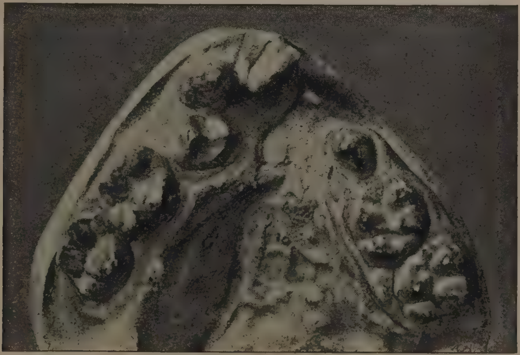

FIG. 136.—Model showing deformity of the arch following the flap operation for cleft palate. (From *Brit. Dent. Journ.*)

Operations for Cleft Palate.—Operations for the treatment of cleft palate are at times followed by considerable disorganisation of the dental arch (fig. 136), which necessitates free extraction and the adoption of dentures to provide a satisfactory masticating surface. A. T. Pitts [1] states that there is a difference in the type of deformity following the method of operation. In the 'flap' operation the deformity is chiefly limited to the side from which the flap is taken, the arch on the side opposite to the flap being usually normal; in the Langenbeck operation, where tension is exerted on both sides of the jaw by the operation, there is a bilateral contraction of the arch, but the degree of deformity is much less than in the flap operation.

(ii) *Indirect Injury* (*causing Displacement usually during Eruption*).

Habits.—Certain habits are contracted by children, such as finger-sucking and lip-sucking, which, if persisted in, will produce deformed arches. In the habit of sucking the thumb or finger, the palmar surface of the thumb is placed against the palatal surface of the first incisors, the mandibular teeth being closed on the dorsal aspect. The pressure exerted causes the maxillary first incisors to protrude, so that the lower lip passes behind them and aggravates the protrusion when the mouth is at rest. At times the thumb or finger is inserted between the teeth in such a manner as to cause the teeth to impinge on the lateral surfaces. Under such conditions, the maxillary incisors and canines are forced outwards and frequently upwards, producing a type of irregularity known as 'open bite.' Sucking of the lower lip may force the mandibular teeth backwards and the maxillary teeth outwards. One has seen a child three and a half years of age with the mandibular incisors forced backwards over the tongue, these teeth being three-quarters of an inch behind the maxillary incisors. Another habit acquired consists in forcing the tongue over the tips of the mandibular teeth and against the maxillary incisors, causing protrusion of the latter teeth.

The prolonged and persistent use of the 'comforter,' or 'dummy,' is a fruitful source of dental deformity. Fig. 137, the models of a child aged five, illustrates the protrusion of the maxillary arch from this cause. The protrusion was well marked when the child was about eighteen months old.

Careful inquiry into the use of the 'dummy' would seem to show that, if its use is persisted in, deformity of the maxillary dental arch will almost certainly result. The extent of the deformity will depend on the frequency with which the 'dummy' is used, the period over which its use extends, and the presence or absence of rickets.

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[1] *Brit. Dent. Journ.*, May 1, 1922.

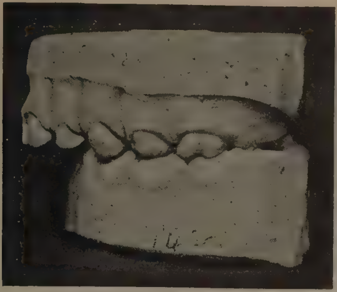

FIG. 137.
(From *Proc. Roy. Soc. Med.*)

The maxillæ and mandible illustrated in fig. 138 show the type of deformity usually seen in children addicted to thumb-sucking. The maxillary incisors project, while the arch of the mandibular incisors is flattened. There is reason to think that in these cases the force on the incisor teeth in the backward

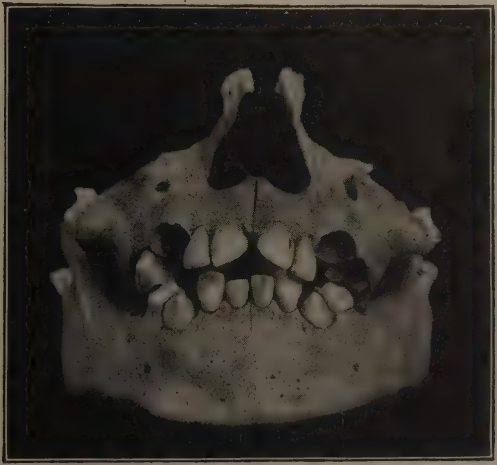

FIG. 138.—Maxillæ and mandible showing protrusion of the maxillary incisors as seen in children addicted to the habit of thumb-sucking.

direction is resisted at the articulation, with the result that the neck of the condyle takes an inward direction, as shown in fig. 139.

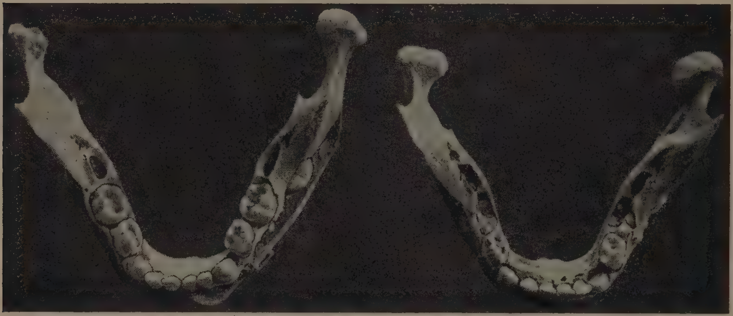

FIG. 139.

Normal mandible.

Abnormal mandible, showing bend in the neck of the condyle.

Results of Cicatrices.—If the soft tissues in the neighbourhood of the jaws have been injured the contraction of the cicatrix may divert the teeth from their normal positions. A case of protrusion of the mandibular incisors which followed an extensive burn of the soft tissues of the neck is shown in fig. 140.

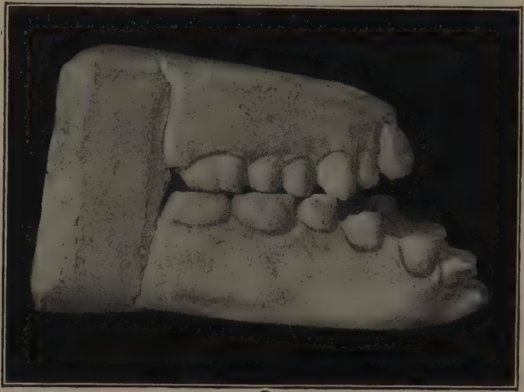

FIG. 140.—Models showing displacement of the mandibular teeth, the result of contraction of a cicatrix. (Tomes.)

Undue Retention of Normal Deciduous Teeth.—The undue retention of a deciduous tooth may divert its successional tooth from its normal path of eruption, so that it comes ultimately to be secondarily misplaced. This is not a rare happening in the maxillary incisor region in particular.

(β) INFECTIVE PROCESSES IN CONNECTION WITH THE TEETH.

(i) *Loss of Tissue from Caries.*—Loss of tissue from caries on the approximal surfaces allows the teeth to close on one another. A forward movement of the permanent molars often occurs from this cause.

(ii) *Infective Processes the Sequelæ of Caries.*—The presence of septic teeth is a common cause of irregularities. Under normal conditions the loss of the deciduous teeth is due to absorption of their roots. When the tissues around a deciduous tooth have been injured consequent on infection and death of the pulp absorption takes place slowly and is often completely arrested. Such teeth obstruct the movement of the erupting tooth, deflect it from its course, and so cause it to erupt in an irregular position.

Similarly granulomata may interfere with the formation and eruption of successional teeth, and cysts also will divert teeth from their normal path of eruption. Inflammatory processes in connection with the deciduous teeth may have the same result.

(γ) CONDITIONS LEADING TO ABNORMAL GROWTH OF THE JAWS.

These consist of (i) errors in feeding and environment, such as rickets in association with adenoids, (ii) errors in manner of feeding, such as faulty appliances for hand-feeding, and (iii) disease, such as congenital syphilis, acromegaly, oxycephaly, progeria and achondroplasia.

By far the greatest number of cases of irregularities of the teeth are the outcome of a diminished growth of the maxilla and the mandible. As regards the most suitable treatment of these cases there is considerable diversity of opinion, which will probably disappear as soon as the pathology of these cases has been established. Before discussing possible causes, it may be advantageous to refer to **the relation of the maxillary-premaxillary suture to irregularities of the teeth.**

An examination of skulls of children suggests that growth of bone at the maxillary-premaxillary suture continues until the eruption of the second dentition is well advanced, and there is reason to believe that the premature closing of the suture is causally related to a crowded arrangement of the incisor teeth.

In the infant this suture is well marked and runs posterior to the foramina leading to the permanent incisor teeth and ends near the margin of the canine socket. Later on the suture tends to be separated by an increasing interval from the incisors. The outer portion of the suture fills in first, the inner portion being often well marked in the skulls of children of from twelve to sixteen years of age.

Palates with well-marked sutures are as a rule wider than those where the suture is ill-defined. A comparison of the inter-canine and inter-deciduous second molar widths of forty-seven specimens gives the following figures :—

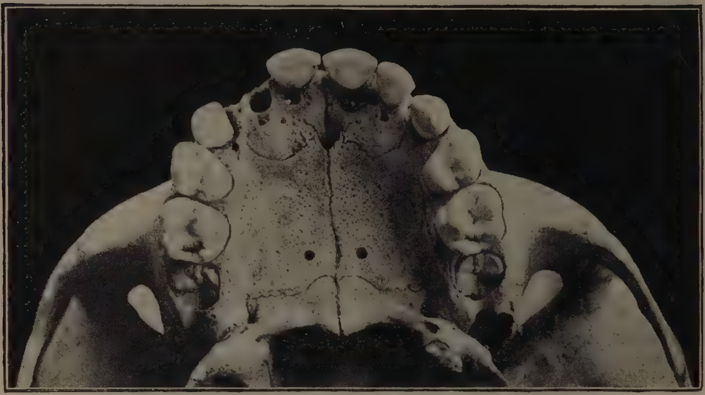

Fig. 141.—Maxillæ of a child about three years of age.

	Inter-canine.	Inter-second molar.
Well-defined sutures (32 specimens) ...	22·5 mm. ...	27·8 mm.
Ill-defined sutures (15 specimens) ...	21·0 mm. ...	26·8 mm.

It will be noted that the difference in the width of the arches is greater between the canines than the molars.

Maxillæ showing well-defined sutures are shown in figs. 141 and 142. In both these specimens the dental arches are well formed and the teeth are not crowded.

In the maxillæ (fig. 143) from a child aged three years two months the outer two-thirds of the sutures are closed, the teeth are curved inwards and are crowded. The canine and molar widths are 19 mm. and 23·5 mm., the normal average being 22·5 mm. and 27·8 mm. The conclusion to be drawn from the examination of dried specimens is that early closure of the suture and crowding of the anterior teeth are definitely related.[1]

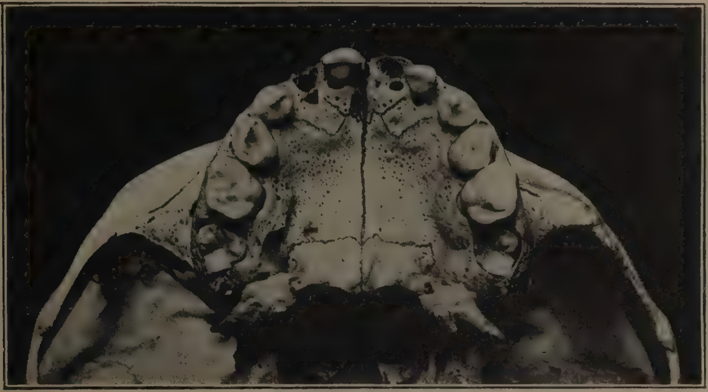

FIG. 142.—Maxillæ of a child aged four years six months. Each premaxilla is developed from two centres.

(i) Errors in Feeding and Environment.—It has been shown that some children are ill-nourished before birth owing to inadequate maternal diet, but that they usually overcome that

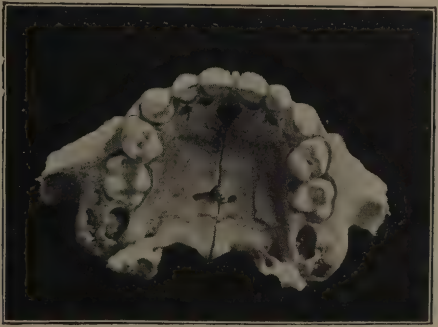

FIG. 143.—Maxillæ of a child aged three years two months with crowded incisors associated with partially closed sutures.

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[1] A fuller account of the relation of the maxillary-premaxillary suture to irregularities in position of the teeth will be found in the *Dental Record*, February 1914.

deficiency if adequately fed afterwards. It stands to reason, too, that if after birth the growing child does not get a well-balanced diet—that is, one containing all the chemical substances including vitamins that the body requires, and in an assimilable form—its body will suffer in some manner. With regard to abnormalities in position of the teeth, that is particularly shown with reference to rickets, a deficiency disease partly caused by a deficiency of vitamin D, and very often associated with adenoids causing nasal obstruction.

The imperfectly calcified and softer bones of rickets are influenced by the extra tension put on them by the cheeks in the process of mouth-breathing, and so become distorted. The grave deformities of the dental arches and consequent crowding of teeth which can be caused in this manner have largely disappeared in the past twenty years with the greater knowledge of the dietetic and physiological needs of small children.

Errors in feeding and in the manner of feeding are frequently coincident, and this tends to exaggerate further the distortion of the plastic bones of rickets.

Nasal obstruction arising from the *presence of adenoids* greatly influences the growth of the maxilla, the effect produced depending on the period of onset and the persistence of the adenoids. The earlier the age at which the adenoids appear the more marked will be the effect on the teeth and palate, and the longer they persist the greater will be the damage.

The Character of the Deformity.—If the mouth of a child of four years of age that was breast-fed and has not had adenoids be examined, the teeth will be found to form a regular arch, with the anterior teeth separated by slight spaces. The vault of the palate in section across the deciduous first molars will be oval-shaped, and, in longitudinal section, will rise with a gentle incline from behind the anterior teeth to the vault (see fig. 144).

In a child that has suffered or is suffering from adenoids, the teeth will be crowded and the muco-periosteum of the palate will present a somewhat puckered appearance (fig. 145); the vault, in transverse section, will be dome-shaped, and, in longitudinal section, will rise somewhat abruptly from the posterior aspect of the incisors, the palate will have the appearance of having been pushed up towards the front, and in some cases the deciduous molars will occlude too far forward.

If the adenoids have not been severe and are removed before the age of six, their effect on the permanent teeth will be to crowd the maxillary incisors, the disturbance ranging from a slight tilt of the second incisors to an irregularity involving the four anterior teeth, but the permanent first molar will erupt in good position and, provided that the deciduous molars are retained for the normal period, the premolars will erupt in normal occlusion. The canines, when in place, will show a slight slope of the roots towards the median line. The only permanent irregularity, therefore, will be the crowding of the incisor teeth.

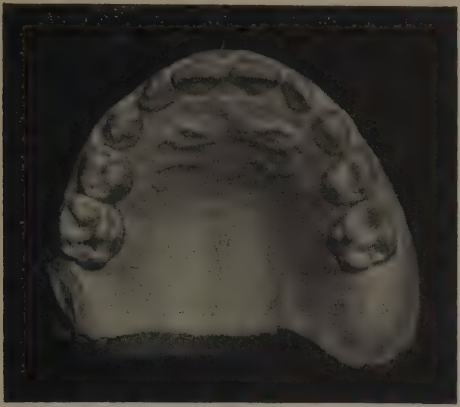

Fig. 144.—The model of a child that was breast-fed and had not suffered from adenoids.

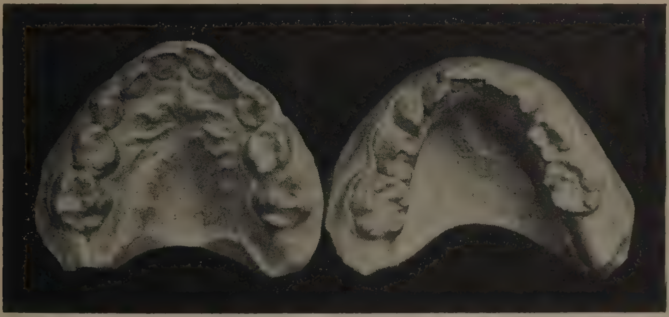

Fig. 145.—Models of a child, aged five years and seven months, that had suffered from adenoids from the age of two years. The puckered appearance of the muco-periosteum covering the hard palate and the crowding of the teeth are well shown.

If the adenoids have been severe and of long duration, an additional effect on the arch will be seen in the region of the permanent first molars, so that these teeth, when erupted, fail to assume a vertical position, and lie with a general slope backwards. This is well shown in fig. 147, the models of a child who suffered from adenoids. It is not difficult to foresee that, as soon as the deciduous second molar is removed, rapid forward movement of the permanent first molar will result in an encroachment on the space for the premolars and an abnormal occlusion.

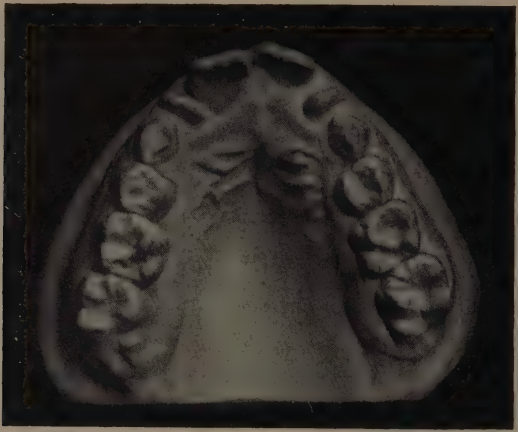

Fig. 146.—Model of the case shown in fig. 145 after the eruption of the incisors. Note the irregular position of the incisors.

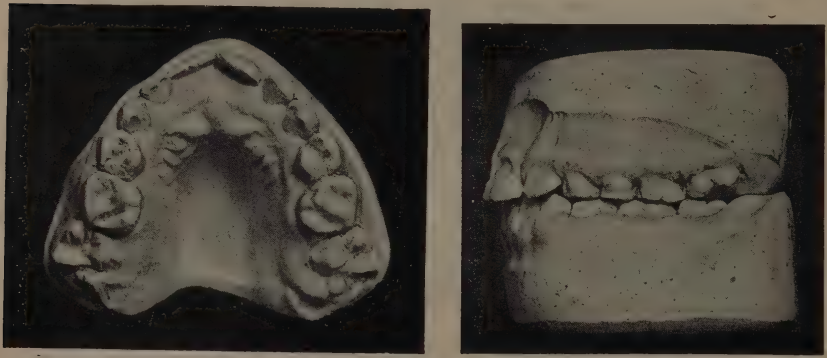

Fig. 147.—Models of a boy aged seven. He had been operated on for adenoids three times. (From *Proc. Roy. Soc. Med.*)

In some cases the growth in the molar region is so much restricted that the permanent first molar erupts in such a way as to lead to absorption of the posterior roots of the deciduous second molar (fig. 148). Under such conditions it is obvious that abnormal occlusion must result and that the space for the premolars will be considerably curtailed.

If the permanent molars do not move forward, the premolars and canines erupt in normal position, and then the only deformity that remains is slight crowding of the incisors. If the permanent molars do move forward, the space for the premolars and molars is reduced and a general crowding of these teeth results.

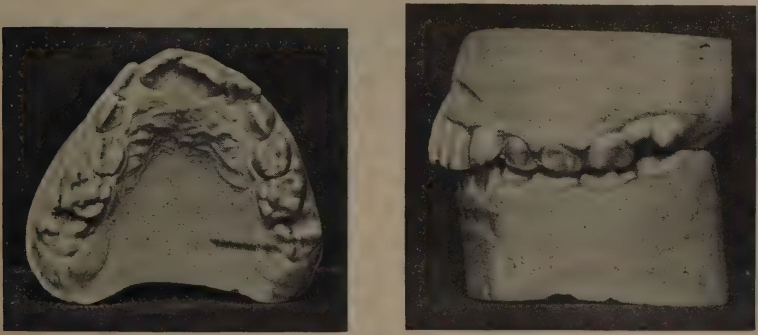

Fig. 148. (From *Proc. Roy. Soc. Med.*)

There would appear to be a slight decrease in the width of the arch in children with adenoids, as compared with those free from adenoids, the difference being slightly less than 1 mm. The subjoined figures would appear to substantiate this view.

	Width between the permanent first molars.	Width between the deciduous first molars or first premolars.
	Breast-fed.	
	A	B
Without adenoids (20 cases) ...	34·85 mm.	26·31 mm.
With ,, (13 ,,) ...	33·80 ,,	25·19 ,,
Difference	1·05 ,,	1·12 ,,
	Hand-fed.	
Without adenoids (34 cases) ...	34·10 mm.	25·61 mm.
With ,, (30 ,,) ...	33·36 ,,	24·90 ,,
Difference	0·74 ,,	0·71 ,,

The width (A) between the permanent first molars was measured from the gum margins immediately in line with the fissures on the palatine aspect of the teeth. The width (B) between the deciduous first molars or first premolars was measured from the gum margins at the point corresponding to the centre of the palatine aspect. The same plan is followed in the measurements shown on pp. 131 and 132.

These measurements are from patients treated in private practice.

With persons who have suffered from adenoids the maxillary third molars often erupt with the occluding surfaces facing backwards and outwards.

Many of the gross lesions of the jaws which are found in association with adenoids are not necessarily the direct result of the adenoids alone. Usually there are other contributory causes, the influence of which will be considered subsequently.

The Factors producing the Deformity.—There is a wide divergence of opinion as to the precise manner in which the deformities of the palate associated with adenoids are produced. It has already been pointed out (p. 84) that the efficient performance of nasal respiration is essential for the full growth of the maxilla. This function is hindered or may be entirely in abeyance when there is nasal obstruction, and the result is that the growth of the jaw is considerably hampered.

H. Campbell, dealing with the subject of nasal respiration, says, ‘When we reflect that a person normally breathes some twenty thousand times through the nose in twenty-four hours, and that the constant flow of air through the nasal passage attracts an abundant supply of blood to the nasal mucous membrane, for the purpose of warming and moistening the air, giving off to it about two quarts of water per diem, it is manifest that the cessation during the developmental period of the respiratory function of the nose must interfere with the proper development of the nasal apparatus, just as early excision of an eyeball tends to defective development of the corresponding orbit.’

The interference with growth would appear to occur mainly in the region of the incisors and the molars. Lack of use of the muscles of the anterior nares, in addition to the loss of stimulus to the nasal mucous membrane, inhibits the growth of the premaxillæ. The restriction of growth from this cause would seem to afford an explanation of the fact that crowded incisors are almost always associated with cases in which adenoids develop during the first six years of life. The want of growth in the molar region is due to the lack of expansion of the maxillary sinus arising from disuse, and the want of growth of the maxilla is correlated with a diminished growth of the mandible.

J. G. Turner[1] is of the opinion that the interference with growth of the maxillæ is not due to disuse of the nose, and, in support of his contention, quotes cases in which tracheotomy tubes have been worn by children for years without any ill-results on the nasal or palatal development. He considers that the cause of the failure of maxillary growth in adenoids is obscure.

The decrease in the width of the arch seen in patients with adenoids may be due to the general want of growth of the maxilla arising from the presence of the adenoids in conjunction with the pressure exerted by the tension of the cheeks in consequence of the open mouth, and the absence of pressure from the tongue.

The measurements quoted on p. 127 show a slight narrowing of the arch in children affected with adenoids. In many of the cases included in the series the nasal obstruction was not sufficient to cause excessive mouth-breathing and the effect of the tension of the cheeks would not be marked; still further, in each case the full complement of teeth was present. In the breast-fed children affected by adenoids there is a greater proportional narrowing of the arch than in hand-fed children similarly affected. At first sight this difference is rather surprising, but the explanation is as follows: The arch of the breast-fed child is broader and the teeth more spaced than in the hand-fed child; pressure brought to bear on the outer surface of the arch forces the teeth inwards; in the breast-fed child the teeth must move a greater distance before mutual antagonism of the teeth occurs than in the hand-fed child, and hence can be explained the greater proportional reduction in the width of the arch in the breast-fed child. These facts support the view that the tension of the cheeks is an important factor in reducing the width of the arch.

It is evident that when once the teeth are laterally in contact the resistance to the lateral pressure is increased, and that any further narrowing of the arch will depend upon whether the pressure of the cheeks is sufficient to overcome the resistance of the arch. If the mouth-breathing is extreme, the effect of the lateral pressure of the cheeks is accentuated and may become sufficient to drive forward the anterior curve of the arch and cause protrusion of the teeth and so produce the saddle-shaped arch which was once common but is now seldom seen.

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[1] *Trans. 17th Int. Med. Congress*, Section of Stomatology, 1913, 57.

If the continuity of the arch is broken by the loss of teeth the narrowing will be accentuated. That the constant tension of the cheeks, and especially the tension of the muscles, does eventually affect the arch is shown by an interesting case recorded by Lambert Lack,[1] in which a child had suffered from unilateral facial paralysis from infancy, in addition to mouth-breathing from adenoids. On the unparalysed side the arch was crushed in, but not on the paralysed side.

A word must be said with regard to the high arch of the palate seen in children suffering from marked adenoids. It has been maintained that this is due solely to the lateral pressure of the cheeks, but that view is open to criticism, as in many cases the narrowing is very slight and does not account for the increased height of the palate. It seems probable that the height of the palate may be due to interference with the growth of the sphenoid and the septum of the nose.

'The hard palate in the infant is normally high arched, and at birth it lies above the level of the Eustachian tube, but later on becomes considerably lower. This alteration is due to the downward growth of the hard palate, so that one factor in the production of a highly arched palate is a lack of development of the walls of the nose, more especially of the sphenoid and the septum.' Any factor such as adenoids will interfere with the growth of the nasal septum, and so retard the proper development of the hard palate and consequently its descent.

To sum up, it would seem probable that the *deformities associated with adenoids and rickets are to be attributed mainly to defective growth of the maxillæ, and that the narrowing of the arch is in a large measure due to the tension of the cheeks*, the severity of the deformities depending upon (1) the degree of adenoids and consequent mouth-breathing, (2) the duration of the adenoids, (3) the loss of teeth from the dental arch, (4) the degree of malnutrition (rickets) present.

The jaws in health preserve their form under stress of mastication, and similarly, when the bone is soft as in rickets and there is corresponding muscular weakness, no change of form occurs. H. K. Hatfield,[2] who examined fifteen children with a clear diagnosis of rickets, found that in the jaw development there was nothing which could be regarded as characteristic of rickets.

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[1] *Trans. 17th Int. Med. Congress*, Section of Stomatology, 1913, 67.

[2] *Dental Items of Interest*, September 1919.

If, however, the functional equilibrium is disturbed by the introduction of an artificial force such as a ‘ dummy,’ or if a change of muscular pressure takes place, as in mouth-breathing, deformity ensues. These altered conditions produce deformities even in healthy bones, but in rickets, owing to softening of bone, the deformities are increased.

(ii) Errors in Manner of Feeding.—There is ample evidence that the method of infant feeding influences the shape of the palate and dental arch.

A series of measurements made of the arches of children free from adenoids gave the results shown below :—

	A	B
Breast-fed	34·88 mm.	26·31 mm.
Hand-fed	34·1 ,, ...	25·61 ,,

The figures recorded above show that breast-fed children have slightly broader arches than those fed by artificial methods. The measurements given relate to patients in private practice ; figures gathered from patients in hospital show a greater difference between the arches of breast-fed and hand-fed children.

To ascertain the comparative breadth of the height of the palate, plaster casts were made of the patients, and these were divided at a point corresponding to the middle of the deciduous second molar and second premolar. Tracings on paper were then made and a line drawn between the gingival edges, and from the centre of this base a line was drawn at right angles to ascertain the height. In the case of breast-fed children the breadth of the palate averaged 2·62 times the height, in the hand-fed children 2·52 times the height.

In hand-fed children there is a greater tendency to abnormal occlusion than in breast-fed children.

It seems quite feasible that this variation in the palates and arches of breast- and hand-fed children is due to the difference between the action of the palate on the teat and the nipple. An infant, when at the breast, takes not only the nipple, but a considerable portion of the areola into the mouth, and the tongue is often seen protruding over the lower lip. In the act of feeding, the mandible is first slightly depressed and then raised so as to squeeze the breast against the palate, and the milk so expelled is then swallowed. The jaws then separate to admit more milk into the flaccid apex of the breast, and the process is repeated. The general effect of this action is to bring the muscular tissue about the jaws into activity, and to widen the palate.

With the modern-shaped bottle the child is held on the left arm and the bottle is kept at such an angle that the milk passes into the teat ; the mandible then exerts a squeezing action and the milk, passing into the mouth, is swallowed. If the bottle is used correctly, very little sucking is performed by the child, the whole act simulating that of the child at the breast ; but there is this difference : the teat is not so tough and resistant as the breast, and the pad in the mouth exerts less extensive pressure on the anterior part of the palate ; still further, the backward force of the breast upon the anterior part of the arch is absent.

The ideal teat has yet to be designed, but it seems obviously desirable that so far as possible the natural action of the nipple should be reproduced, and to this end teats for bottles should be made flat and broad at the base, a little longer than those at present used and of a tougher material. Now that so many children are hand-fed the matter is of considerable national importance.

The modern feeding-bottle is, however, a great improvement on the old tube feeding-bottle. The teat of the old tube feeding-bottle is of a narrow tube-like type from 1 to 1½ inch in length. In taking the milk from this bottle, the tongue is close around the teat and the act of suction is brought into play. The mandible is not brought into use, and the whole action of the muscular tissue of the cheeks is towards the median line, and not away from it as it is when the child is breast-fed. The action tends to narrow the palate instead of spreading it. The average measurements of the arches of forty-one patients who had used the tube-bottle were as follows :—

A	B
32·14 mm.	23·05 mm.

These figures, compared with those of hand-fed by modern feeding-bottles, show a marked diminution in width, especially in the premolar region.

(iii) The Effects of Disease on the Growth of the Jaws.

Congenital Syphilis.—It is common, but not constant, in cases of congenital syphilis to find that there is a lack of occlusion of the teeth in the incisor region ; apparently this is due to a deficiency of growth of the premaxilla (see p. 47 *et seq.*).

Acromegaly.—In this condition growth recommences in certain parts of the body, the mandible being one of the parts affected. The ramus elongates, there is growth along the alveolar border, at the chin, and also at the lower border of the bone. These changes are diagrammatically shown in fig. 149, the mandible of a normal person being superimposed on that of three acromegalic mandibles.

A. Keith,[1] who has given considerable attention to the subject, considers that the increased growth is due to over-action of the pituitary secretion. ‘It somehow sensitises the osteoblasts of the masticatory apparatus—not only wakens up growth in them, but renders them highly susceptible to the mechanical influences to which they are subject when in action.’

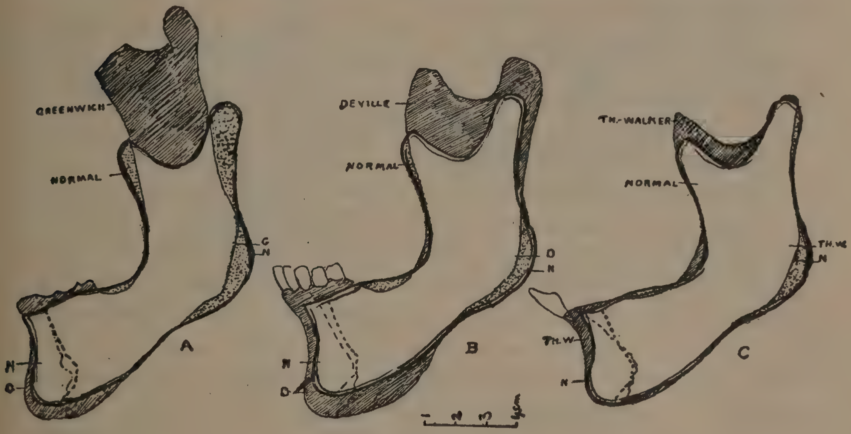

Fig. 149.
(A. Keith.) (*Dental Record.*)

Oxycephaly.—In the condition known as oxycephaly the maxillæ are poorly developed, the length of the maxilla being reduced in proportion to the anterior-posterior diameter of the skull. The maxillary sinus is usually rudimentary. The mandible is not affected.

Morley Fletcher[2] thus describes the jaws in a case of well-marked oxycephaly: ‘The superior maxilla was shortened from before backwards. The hard palate formed a narrow, very pointed arch. The second and third molars were absent in the upper jaw and could be made out in the radiogram lying above on what appeared to be rudimentary antra. Lower jaw underhung teeth normal in number.'

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[1] *Dental Record*, xxxiii, 769.

[2] *Quarterly Journal of Medicine*, 1911, iv, No. 15.

In oxycephaly there is synostosis of the coronal and sagittal sutures and this leads to a lack of growth of the maxilla. There is some evidence that the changes are related to an alteration or abnormal development in the pituitary body.

PROGERIA.—This interesting condition was first described by Hastings Gilford. It is a case of delayed or arrested growth—infantilism—to which is added senilism, a term used to designate prematurely senile changes.

The skeletal changes in a case of progeria have been fully described by A. Keith.[1] The condition of the jaws and teeth is most instructive because it seems to shed light on the problem of the crowded mouth. The palate is very little larger than that of a child at birth, the length being less reduced than the width. On the other hand, the teeth are as advanced in formation as one would expect to find in an individual of eighteen years of age, but are placed in an irregular manner. The second incisors and third molars are absent and the canine unerupted. In the mandible the second incisors are absent, the premolars are embedded in the bone, the first molars have been destroyed by caries, and the second molars are erupting from the inner aspect of the coronoid process. The third molars are absent (figs. 150 and 151).

ACHONDROPLASIA.—In this disease there is a slowing down or arrest of bone growth at epiphyseal lines and, owing to synostosis of the bones of the base of the skull, the maxilla may be deformed. The malformation consists in a retraction of the face and a tilting upwards of the anterior part of the palate.

The manner in which the teeth and jaws are affected in cases of acromegaly, etc., where the disease is well marked, is indicated above. It is probable that these diseases are frequently present in a lesser degree, and that with further knowledge they will be found to be a more common cause of irregularities of the teeth than has hitherto been suspected.

(δ) NEOPLASMS.

The occurrence of a neoplasm can displace teeth from their normal positions ; if they are benign they may divert unerupted

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[1] 'Progeria and Ateleiosis,' *Lancet*, February 7, 1913.

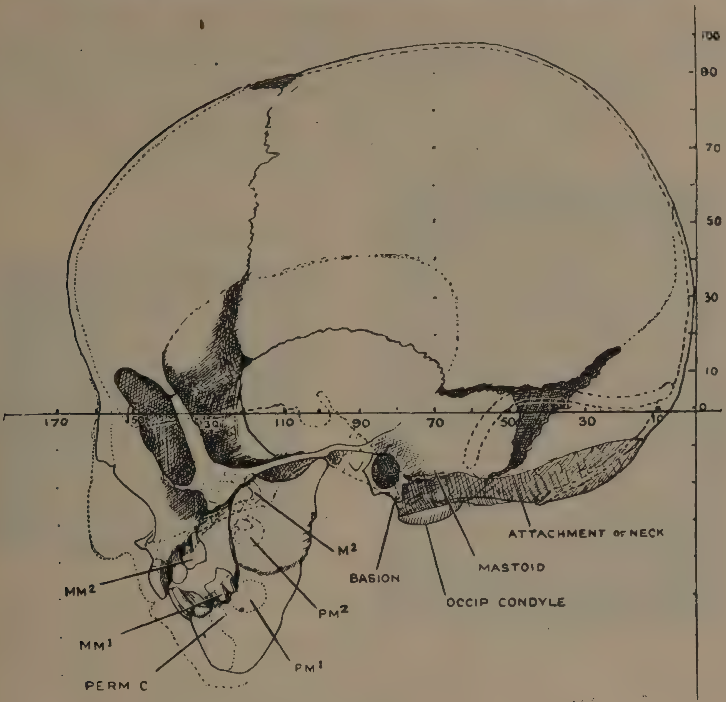


Fig. 150.
(A. Keith.) (*Dental Record.*)

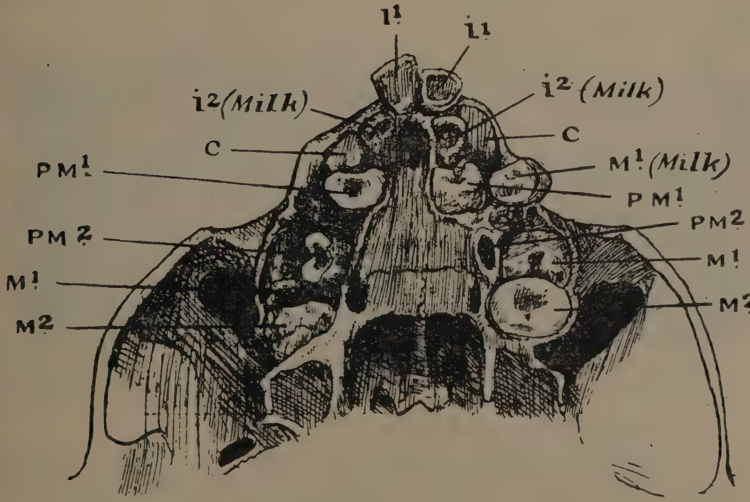


Fig. 151.
(A. Keith.) (*Dental Record.*)

teeth from their path of normal eruption or displace them when erupted, and if malignant they may invade and destroy the germs of unerupted teeth or erode their attachment and support when erupted, so bringing about their loss. By comparison, the loss of teeth under these circumstances is a secondary matter.

(ε) PHYSICAL CAUSES OF UNCERTAIN ORIGIN.

Teeth which only partly erupt or which remain completely unerupted constitute abnormalities in position. No exact knowledge is yet available of the factors which cause this to occur when ample room is available for their eruption ; this matter and the etiological factors in so far as they are known are discussed in Chapter I.

(2) OCCLUSAL (ANGLE'S) CLASSIFICATION.

This classification must be regarded only in the light of occlusion as it affects the position of the teeth, and in particular with the occlusion of the permanent first molars, it is only true as regards the antero-posterior positions of the teeth (see p. 84).

The maxillary permanent first molar is regarded as the 'fixed point' in this classification. A case of normal occlusion at different ages is illustrated for comparison in figs. 152 to 158.

Class I.—The permanent first molars are in normal medio-distal occlusion with each other, the anterior teeth are those which most often are in malocclusion, they may show a general crowding or it may affect individual teeth only (see figs. 159 to 162).

Class II.—The mandibular permanent first molar is in postnormal medio-distal occlusion with the corresponding maxillary tooth. This class is subdivided into two divisions either of which may be unilateral or bilateral.

> Division I.—The arches are narrowed in width, especially in the region of the canines and premolars ; this forces out the maxillary incisors and the mandibular arch is postnormal to the maxillary arch (see figs. 163 to 166).
>
> Division II.—In this type the arch may not be much narrowed, but crowding of the anterior maxillary teeth is the result of too small an arch in which the first incisors are

placed on a plane posterior to the second incisors, which in a typical case are very prominent; the canines also are in line with the second incisors (see figs. 167 to 170).

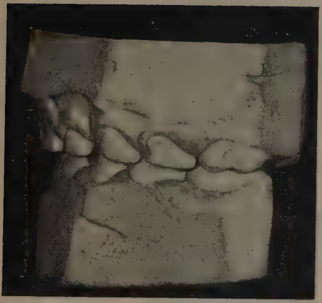


FIG. 152.—Age 4.

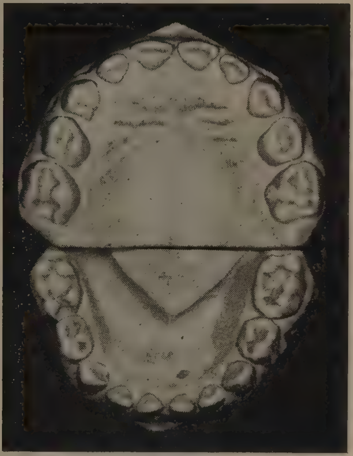


FIG. 153.—Age 4.

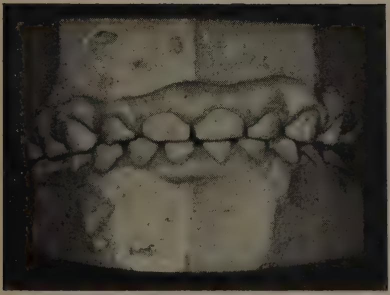


FIG. 154.—Age 4.

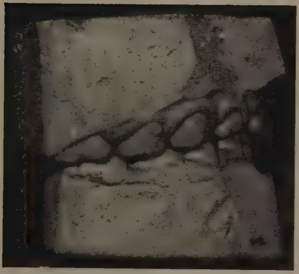


FIG. 155.—Age 4.

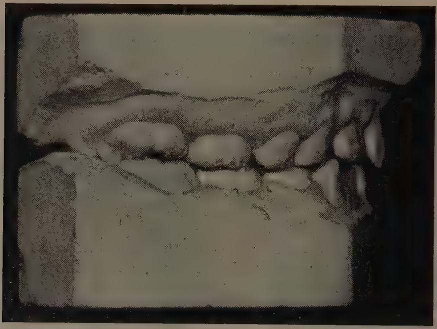


Fig. 156.—Age 9–2.

Figs. 155 and 156 show the increase in length of the arch from age 4 to 9–2.

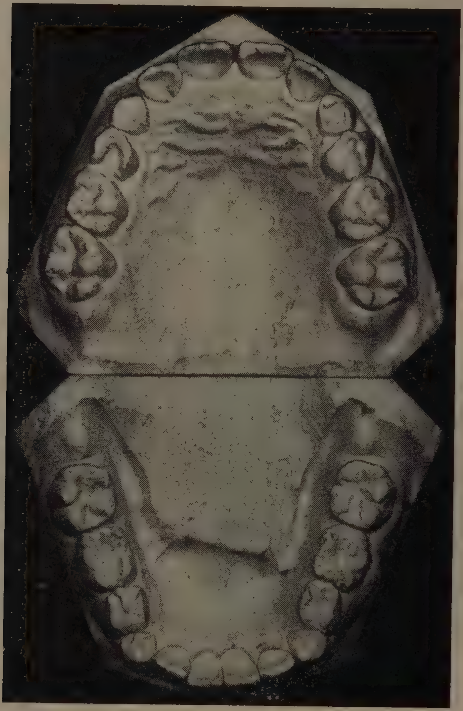


Fig. 157.—Age 9–2.

The width of the upper arch, measured from the occlusal sulci of $\frac{e \mid e}{\mid}$ is 2·2 mm. wider than the same arch in fig. 153. This increase is the result of growth only (no treatment).

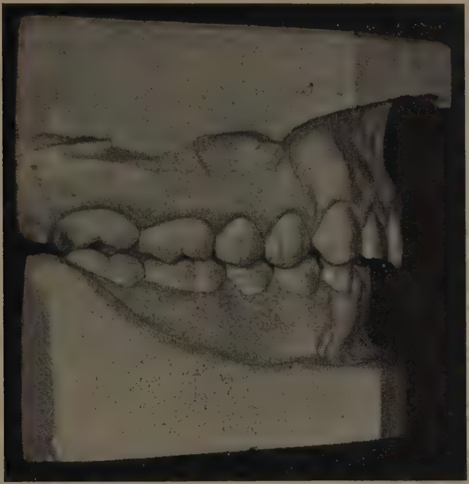


FIG. 158.—Age 20–1.
The same case as shown from figs. 152 to 157. There is slight excess of overbite in these models.

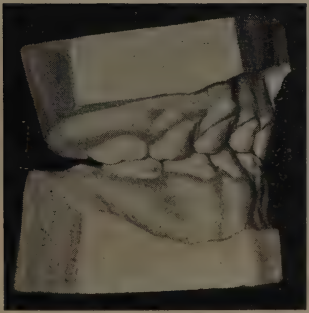


FIG. 159.—Age 4.

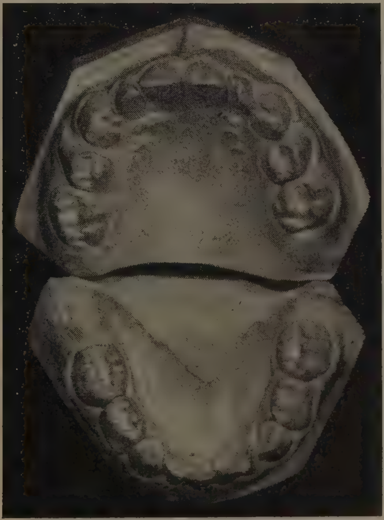


FIG. 160.—Age 4.

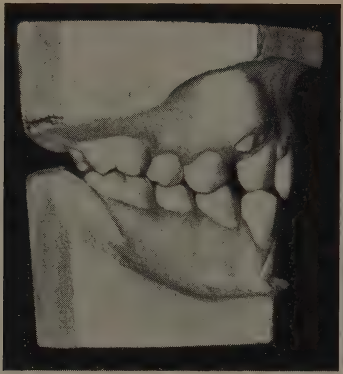


FIG. 161.—Age 12.

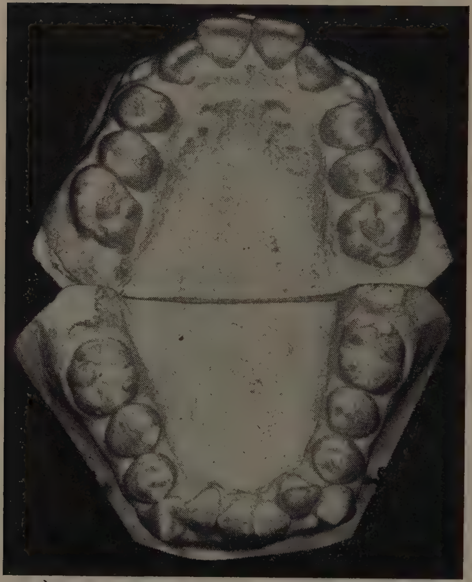


FIG. 162.—Age 12.

FIGS. 159 to 162.—A Class I case.—Models of the same child at 4 and 12 years of age. There is slight crowding and rotation of the incisors in the deciduous dentition and no spaces. In this case at 12 years of age the canines are almost entirely shut out of the arches, the irregularity appearing much worse than in the deciduous dentition; this is because the deciduous arch should consist of teeth and spaces, whereas in the permanent one it usually consists of teeth only; so, a deciduous arch with the teeth in good alignment and no spaces constitutes an abnormality. There was no treatment.

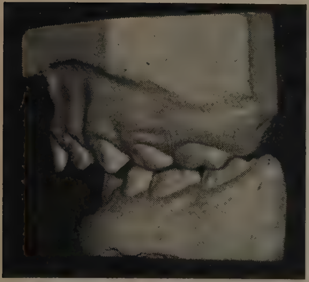


FIG. 163.—Age 4–7.

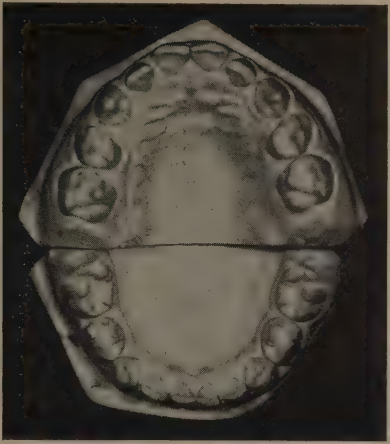


FIG. 164.—Age 4–7.

FIGS. 163 and 164.—Class II, Division I.—Models of the same child. The mandibular incisors have a slightly flattened appearance, probably due to the habit of lip-sucking.

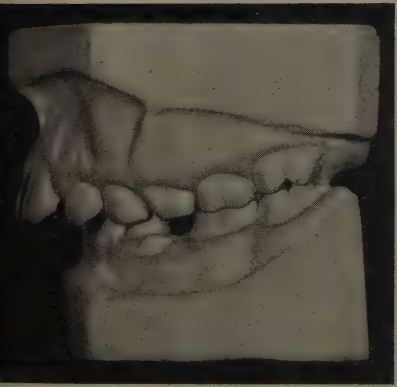


FIG. 165.—Age 9–2.

FIGS. 165 and 166.—Class II, Division I.—Models of the same child. Postnormality nearly one cusp. There has been a little forward drift of the mandibular molars on account of the premature loss of the deciduous first molars, making the postnormality appear less than it actually is.

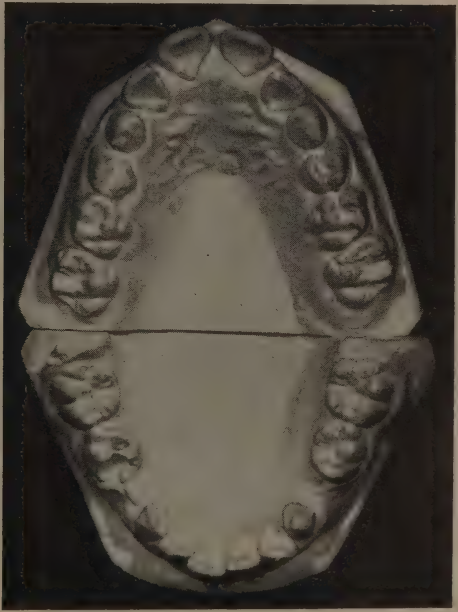


FIG. 166.—Age 9–2.

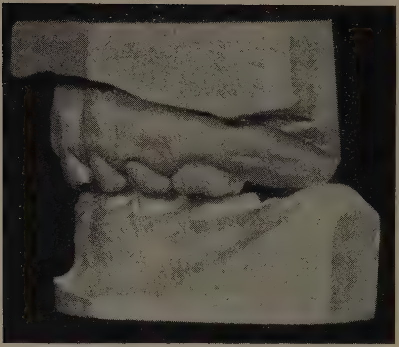


FIG. 167.—Age 7–5.

FIGS. 167 to 170. A Class II, Division II case.—Models of the same child at ages 7 and 11. The maxillary deciduous incisors, instead of being spaced, are even crowded, as evidenced by rotation and imbrication ; the maxillary left deciduous molars are buccal to the mandibular ones. The appearance of the teeth might be considered satisfactory to a lay person. The permanent dentition shows severe crowding of the maxillary incisors, the second incisors being apparently in marked buccal position, but actually the first incisors are lingually placed, the mandibular arch being postnormal. The molar occlusion on the left side has improved bucco - lingually. Treatment should have been undertaken at the time the models in figs. 167 and 168 were made, so that the maxillary incisors might erupt or easily be guided into good positions. There was no treatment.

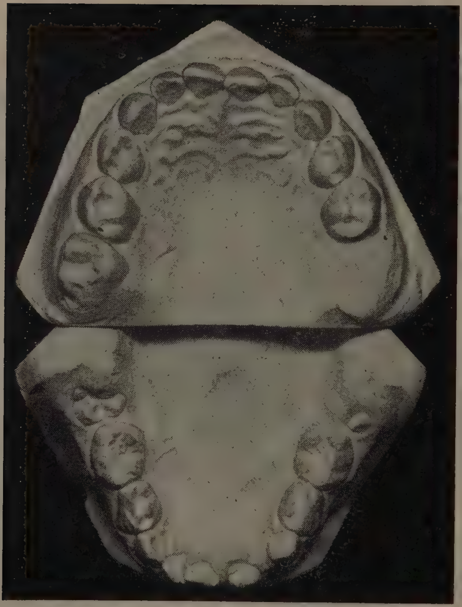


FIG. 168.—Age 7–5.

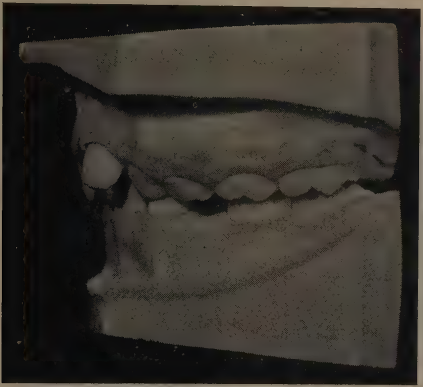


FIG. 169.—Age 11.

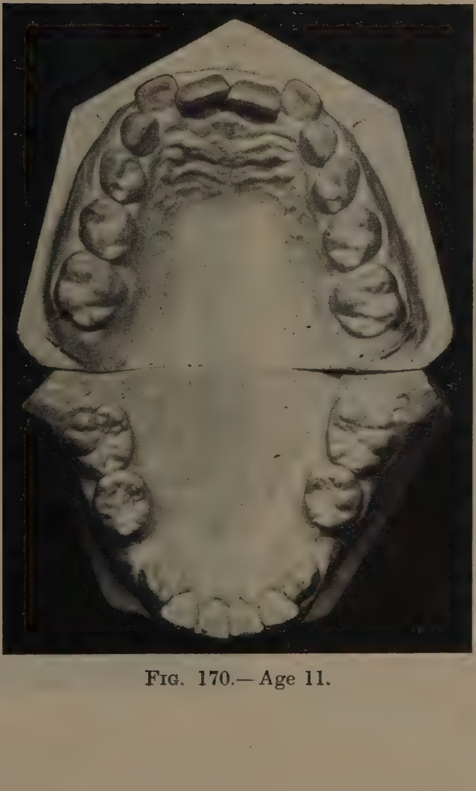


FIG. 170.—Age 11.

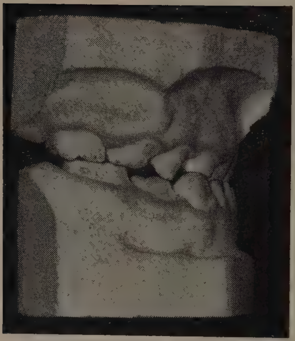


FIG. 171.—Age 7–3.

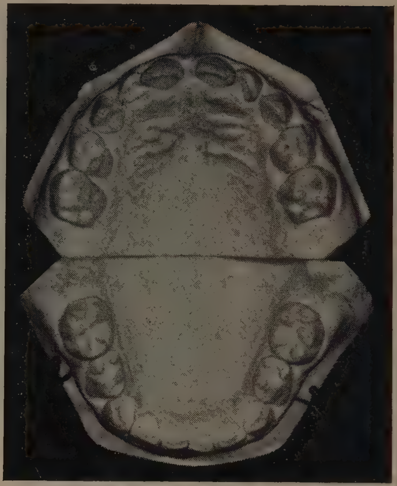


FIG. 172.—Age 7–3.

FIGS. 171 and 172. A Class III case at age 7–3.

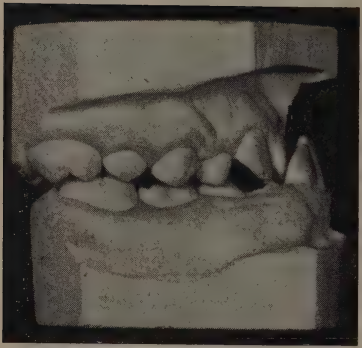


FIG. 173.—Age 9–10.

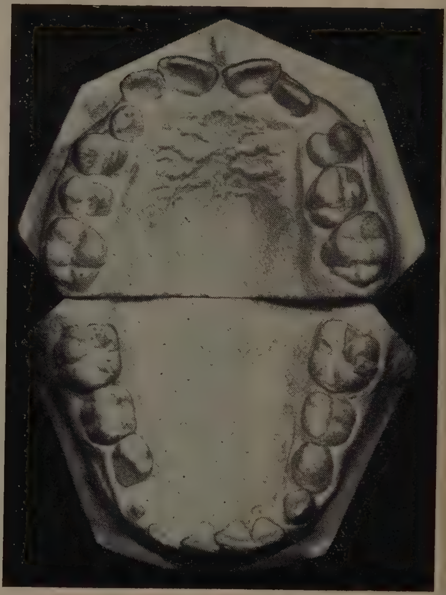


FIG. 174.—Age 9–10.

FIGS. 173 and 174. A Class III case at age 9–10. Excessive overbite has caused a very flattened arrangement of the maxillary incisors.

Class III.—The mandibular permanent first molar is in prenormal medio-distal occlusion with the corresponding maxillary tooth, and in a typical case the mandibular incisors are in labial occlusion to the maxillary incisors. The condition may be hereditary or due to local causes or habits or a bite of convenience (see figs. 171 to 174).

This method of classifying is convenient as a means of rapidly conveying a mental picture of the physical appearance of any given case.

Such abnormalities as open bite, those due to teeth in excess of the normal number and others are omitted from this classification, which does not take etiological factors into account or afford any guide to treatment of such cases.

CHAPTER V

Abnormalities in Position of the Teeth.—Treatment.

Treatment in General—The Movement of Teeth by Mechanical Appliances—The Movement of Teeth by Surgical Methods.

(A) TREATMENT IN GENERAL.

(1) Prophylactic.—Our knowledge of the causes of irregularities of the teeth is as yet far from complete, but year by year investigation discloses fresh facts which assist us in removing or ameliorating those causes, and it is important, therefore, that the student should keep closely in touch with the results of recent research.

It is of primary importance that breast-feeding should be insisted on wherever it is practicable, but if hand-feeding is necessary the food should be administered for preference by means of the spoon or feeding-cup. The use of the comforter should be strongly deprecated. Every care should be taken to prevent caries of the deciduous teeth. A diet requiring plenty of mastication is essential, and proper nasal breathing should be assiduously encouraged. Should nasal obstruction be caused through the presence of adenoids, or from any other condition, treatment should be accorded as speedily as possible, in order that proper nasal respiration may be restored. If the teeth of a breast-fed child are maintained in a healthy and efficient state and nasal breathing is promoted, there is little danger of irregularities arising during the second dentition.

Careful supervision of the mouth during the period of dentition will go far to prevent or, at any rate, to ameliorate irregularities.

Early treatment of caries in the deciduous molars will often prevent their premature loss, while the timely removal of a deciduous tooth will often prevent a permanent tooth from assuming an abnormal position. Special attention should be bestowed upon the permanent first molars. Any cavities which appear should immediately be filled. If caries does appear on the anterior surface of the permanent molar, the advisability of removing the deciduous second molar and making the surface of the permanent molar self-cleansing must be considered. The successful preservation of the permanent first molar far outweighs in importance the slight moving forward of the tooth which might result from the removal of the deciduous second molar. To secure good results, children's teeth should be examined at least three times a year, and the necessity for constant supervision should be impressed on the parents.

(2) Remedial.—Whenever there is doubt as to the best method of treatment, it is advisable to take **models of the mouth,** to be studied in connection with a chart on which the condition of the teeth has been carefully noted. In this way points often become apparent which would be missed by a mere examination of the mouth. Where a case comes under observation at an early stage, it is advisable to take models periodically in order that the development of the mouth can be watched and perhaps some light gained as to the best method to pursue in the treatment.

The teeth must be carefully examined with special reference to the following points :—

(*a*) The general character of the teeth.

(*b*) The presence and extent of caries, especially on the approximal surfaces.

(*c*) The directions of the roots of the teeth. (The value of this point is referred to on pp. 222 and 223.)

(*d*) The occlusion of the teeth.

The last point is of special importance if extraction is indicated. Many of the indifferent results which follow treatment by extraction are due to the removal of teeth without regard to occlusion.

The teeth have a natural tendency to move from their normal positions in the mouth, termed **'travelling of the teeth,'** and in connection with this movement the 'bite' or occlusion of the teeth plays an important part. If a normal articulation (fig. 175) be examined, it will be observed that the opposing teeth present inclined planes to one another, and this is particularly noticeable in the premolar region. With each act of mastication pressure is brought to bear upon these surfaces, and as long as the resistance remains equal in all directions the tooth retains its position.

Remove this resistance, wholly or in part, and the force exerted by the muscles with each act of mastication will tend to force the teeth in the direction of least resistance. Take, as an example, the case diagrammatically illustrated in fig. 176. The permanent first molars have been removed. Each time the posterior plane of the mandibular second premolar strikes the anterior plane of the maxillary second premolar the tendency will be to drive the latter tooth in the direction of least resistance, namely, backwards. When once this tooth has travelled backwards, the resistance to the movement of the mandibular second premolar in the posterior direction is removed, so that the force of the posterior plane of the maxillary first premolar striking the anterior plane of the mandibular second premolar will tend to drive the latter also in a backward direction. In like manner, the posterior plane of the mandibular first premolar will tend to drive back the maxillary first premolar. In addition to the power derived from the muscles of mastication, the force exerted by the lips, tongue, and the process of eruption are important factors in producing the natural movement of teeth. The lips exert force in a backward direction, the tongue in a forward direction. When employing extraction

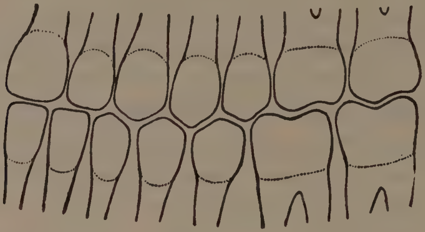


Fig. 175.

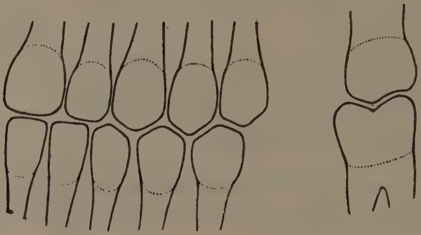


Fig. 176.

for the correction of an irregularity, an endeavour should be made to 'unlock' the bite. This will be more easily understood by a reference to fig. 177. For the sake of argument we will suppose

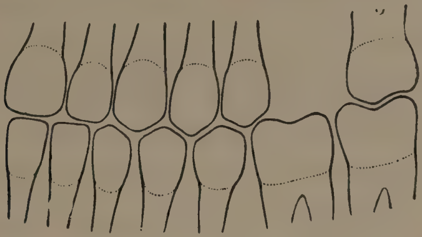


FIG. 177.

that the maxillary canine has erupted externally to the arch. To provide space the maxillary permanent first molar only is removed. The bite will remain locked—in other words, the second premolar will require mechanical means to move it in a backward direction, and even then will occlude with the molar—an unsatisfactory condition. Mechanical means will also be required to retract the first premolar. If the mandibular permanent first molar is removed as well as the maxillary, mechanical methods can be avoided to a very great extent, and there will be less interference with the efficiency of the bite.

When removing teeth in both maxilla and mandible, opposing teeth should, if possible, be selected. A glance at the diagram (fig. 178) will show the disadvantage of not following this course.

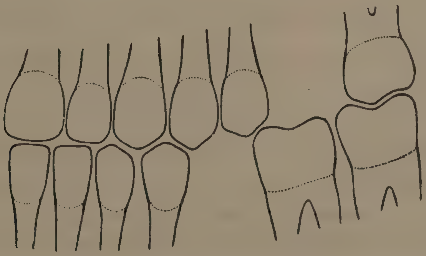


FIG. 178.

In the maxilla the permanent first molar has been removed, and in the mandible the second premolar. There is considerable destruction of the masticating area and, moreover, mechanical methods must be used to retract the teeth. Removal of the maxillary and mandibular second premolars, or the maxillary and mandibular permanent first molars, would have interfered far less with the masticating area, and would have unlocked the bite and allowed the teeth to travel backwards unaided by mechanical means.

In employing extraction the desirability of maintaining the **teeth in their proper relation to the median line of the face** must, for æsthetic reasons, be borne in mind. Any deviation to one side or the other in the direction of the incisors causes an appreciable disfigurement, and, in the case of girls, this is important from an æsthetic point of view. The centre of the mouth is likely to shift when the bite is unlocked on one side and not on the other. Take, as an example, a case where it has been necessary to remove the maxillary first premolar on the left side. The posterior resistance to the canine and second incisors being removed, the pressure of the lips tends to drive all the anterior teeth to that side and so cause the centre to shift and the arch to become flattened on that side. An example is shown in fig. 179. The patient's appearance has suffered considerably. Removal of the maxillary right first premolar as well as the maxillary left first premolar would have prevented the disfigurement because, the posterior resistance on both sides being removed, the lips would have forced back the anterior teeth equally on both sides. Removal of one maxillary second premolar will not cause the centre to shift because the bite will be locked by the maxillary first premolar occluding with the mandibular premolars, but removal of the fellow tooth in the mandible will withdraw the posterior resistance and permit the first premolars—maxillary and mandibular—to shift back and so produce an alteration in the centre of the mouth. Other examples could be quoted. Removal of the maxillary and mandibular right permanent first molars and the maxillary left permanent first molar would cause the centre to shift towards the

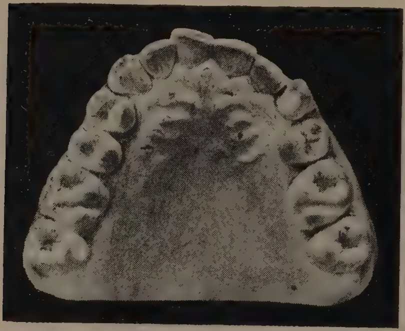


Fig. 179.

right side because the bite is locked on one side and not on the other.

Radiograms of the teeth are a necessity in cases where doubt exists as to the directions of the roots of teeth, or as to the presence or absence of unerupted or supernumerary teeth.

In any case where there is a question of treatment by mechanical methods the **general health and temperament** of the patient must be considered. With weak, nervous children mechanical treatment should, for obvious reasons, be avoided if possible. Again, in all cases requiring mechanical treatment the thorough co-operation of parents and patients is most essential to success. Orthodontic apparatus, unless constantly worn and properly attended to, does more harm than good. If the practitioner has reason to believe that his instructions will not be faithfully carried out, prolonged mechanical treatment should be avoided.

Other points to be considered are the **age** and the **sex** of the patient. With regard to the former it should be remembered that the difficulty of moving and retaining the teeth in a new position increases with the age of the patient. As a rule, it is not advisable to attempt correction of irregular teeth by mechanical means in adults. The older the patient the more difficult the teeth are to move and retain in a new position. Again, the older the patient the greater the danger of starting pathological changes in the parodontal tissues.

In deciding on the method of treatment, attention must be given to the **facial expression and type of face.** For example, a narrow arch may be accompanied by a narrow face with a small mouth and correspondingly small features, all of which are in harmony. Expansion or spreading of the arch under such conditions might mar the character of the face.

The removal of the canine usually produces an ugly flatness of the upper lip and allows the angles of the nose to sink ; and the increased noticeability of the naso-labial fold thereby produced later on gives a premature appearance of age.

In cases where, with protrusion of the maxillary teeth, the lower lip is unduly flat, but the mandibular teeth are crowded, with perhaps the canines slightly prominent, an attempt to regulate the canines by bringing them into line will tend to increase the flatness of the lower lip and to accentuate the protrusion of the maxillary teeth.

(B) MOVEMENT OF TEETH BY MECHANICAL MEANS AND REGULATING APPLIANCES.

The movement of teeth by mechanical means is accomplished by the application of certain forces which act from a fixed base known as '*the point of delivery*.' The resistance at the point of delivery, or anchorage, must be greater than that of the tooth or teeth to be moved. This is an essential condition which is frequently overlooked.

The point of delivery is usually obtained from the resistance of teeth either by means of a well-fitting plate or by embracing two or more teeth with a clamp or band, the force to be used being attached to the plate or band. In a few forms of apparatus the occiput is utilised as the point of delivery. Regulating appliances are thus divisible into two main groups: (*a*) removable, (*b*) fixed.

(*a*) **Removable Appliances.**—The successful working of a regulation plate depends mainly upon accurate fitting, and great care should therefore be taken in obtaining models of the mouth. The best material for the plate is vulcanite. The posterior teeth should sometimes be capped, as the real point of delivery is obtained from the teeth, and by capping them a much firmer hold can be obtained. Plates must be carefully adjusted to the bite of the opposing teeth. The plate should be tried in the mouth and the bite adjusted by the aid of a proper articulator. It is advisable to allow the lower teeth to bite well 'home' into the vulcanite, as the plate is then kept more firmly in position, and, when the mouth is closed, the opposing teeth also act as part of the point of delivery.

The Forces employed for Moving Teeth.—(i) **The screw** is perhaps the most powerful. The types suggested by E. H. Angle are neat and efficient.

The screw must be adjusted at regular fixed intervals, only a slight degree of force being applied at each visit. The force exerted by a screw is not continuous, but this is evidently no disadvantage, as the results obtained from screw force in regulating teeth are entirely satisfactory.

(ii) **The Wedge.**—Wedges may be made by fitting vulcanite pegs into cone-shaped holes drilled into the plate, the peg being left sufficiently long to prevent the plate being forced at once into place. The pegs may also be made of hickory wood, the length of the grain being parallel with the direction in which it is desired to move the tooth, though this, being absorbent, is not so clean. This method is useful for moving incisors out. Although this method is called wedging it is really a plate acting on an inclined plane, the plane being the sloping surface of the tooth it is desired to move. The peg is so arranged with a sloping surface that at first it presses on the tooth near the cutting edge, and as the plate is driven up into place by the mandibular teeth so the tooth is forced forwards. It is important that the mandibular incisors should occlude with the plate and so increase the force transmitted to the teeth to be moved.

(iii) **The inclined plane** is most usefully employed when it is necessary to move forward three or four maxillary incisors. For this purpose a plate is made capping the mandibular teeth and an inclined plane is fixed to the plate, so that when the mouth is closed the teeth to be moved impinge on the sloping surface and are thus driven forward. The inclined plane must be used with care, as the soft tissues may be injured if the teeth are moved too quickly.

(iv) **Elasticity.**—The force obtained from the elastic properties both of rubber and metals, especially the latter, is most useful for mechanical regulation of teeth.

(*a*) *Rubber.*—This material is generally used in the form of small bands. There is always some difficulty in retaining bands in position on the teeth, the tendency being for the bands to slip towards the necks of the teeth and cause inflammation. This can be overcome by cementing to the teeth metal bands with hooks, spurs, or studs attached. The rubber bands require frequent renewal and are somewhat uncertain in their action.

(*b*) *Stainless Steel Wire* is now often used in orthodontic work and possesses advantages which its name implies. It is not easy to attach auxiliary springs to it, though this can be done by means of an electro-welder, and it can now be done by special ‘ soldering ’ means.

(*c*) *Platinised Gold Wire* is now often used for orthodontic work. It is very elastic, though not so much so as steel wire. It is used in gauges about 20 (American) for arches and 23 for auxiliary springs.

In figs. 180 to 187 some methods of employing a highly resilient wire such as platinised gold or stainless steel are shown.

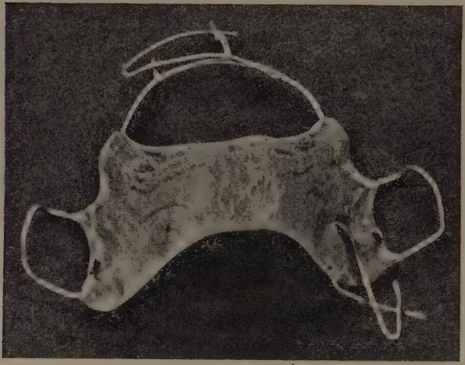


FIG. 180.—Plate to move |12 labially and 6| buccally; the free end of spring |12 may be extended to coil round the lingual wire beyond the attachment of the spring so that any tendency to displacement is lessened. Palatal view.

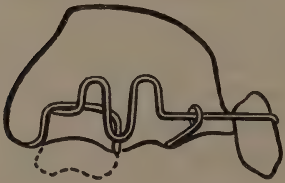


FIG. 181.—Plate for retracting maxillary permanent canines. Lateral view.

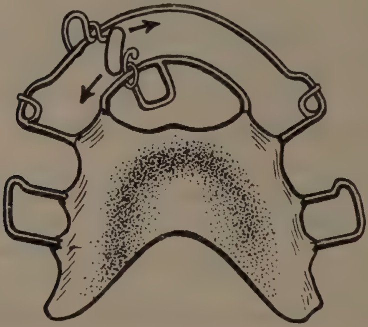


FIG. 182.—Plate to rotate 2|, lingual spring to move distal corner disto-labially and labial spring to move medial corner medio-lingually Lingual view.

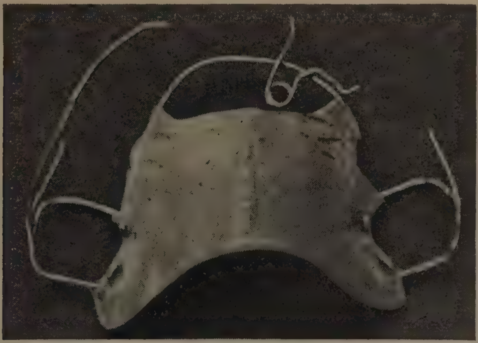


Fig. 183.—This plate has moved 1| medially, and has still to guide 2| medio-labially, move |2 disto-lingually and retain 5|5 lingually. Palatal view.

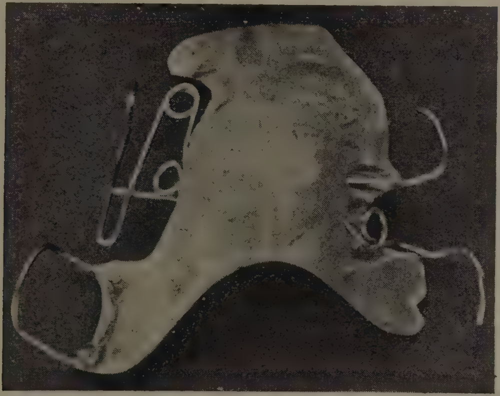


Fig. 184.—To move 6| distally and 4| medially (these springs also act as clasps) and |45 buccally. Palatal view.

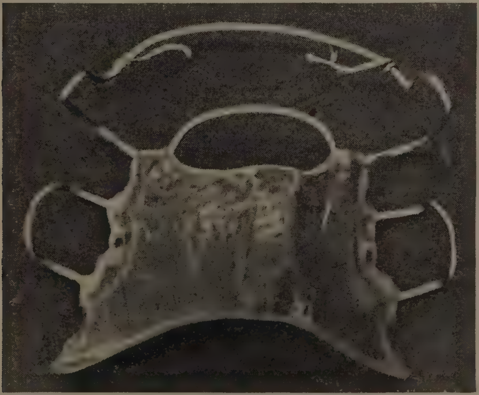
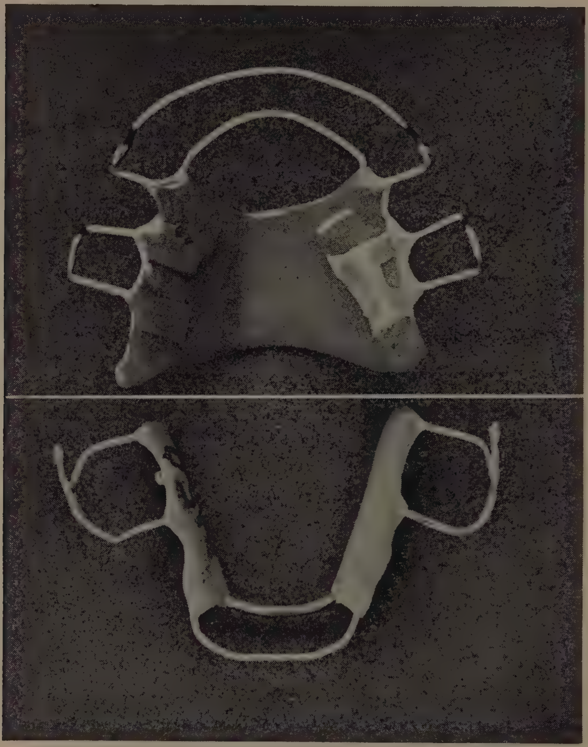


Fig. 185.—Springs to move maxillary second incisors disto-lingually; a greater length of spring than that shown is desirable. Spur to keep spring for 2| in position. Palatal view.

(*b*) **Fixed Appliances.**—With fixed types of appliances the ‘point of delivery’ is obtained by banding two or more teeth and fixing to the bands the appliance or arch from which the force is delivered. For the following description of fixed appliances we are indebted to Harold Chapman, who has made a special study of this branch of orthodontics.



Figs. 186 and 187.—Retention plates: wire is substituted for vulcanite round the anterior teeth: intermaxillary traction may be worn with these plates at night (after continuous use of intermaxillary traction with fixed appliances). If the wire at the necks of the teeth lingually were cut it could be used as a spring. Lingual views.

The form of fixed appliance which finds favour with the majority of practitioners consists of bands, the anchor bands, attached to certain teeth, the anchor teeth, the bands being connected by a wire which is placed on either the lingual or labial or both aspects of the teeth. This curved piece of wire is generally known as the 'arch.' In young children the anchor bands should be fixed to the deciduous second molars, in older children to the permanent first molars.

The anchor bands, which should be made of platinised gold or stainless steel, may be fitted directly to the teeth or they may be made on models of the teeth. Tubes are soldered or welded to the bands to afford attachment for the arch. The arches, if of precious metal, should be made of platinised gold with a high percentage of platinum to make a springy alloy which will retain its resiliency after it has been heated.

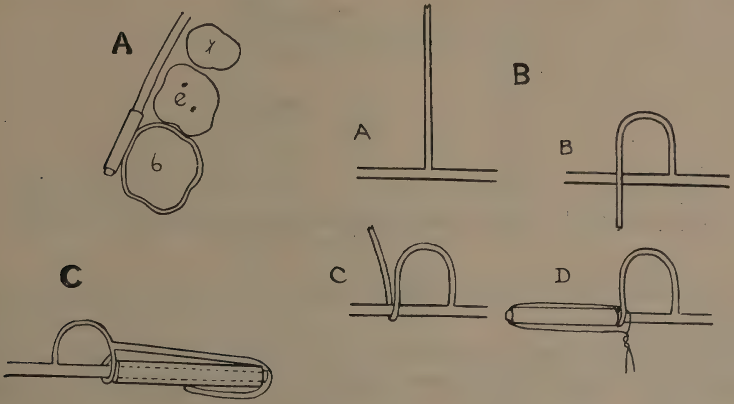


FIG. 188.

A. Shows the attachment and alignment of the horizontal buccal tube to the anchor band; the tube is not attached 'solidly' to the band, the two extremities being free; the niche so formed is used distally to hold the ligature or soft lock-wire; if the niche does not exist, the arch must extend through the tube to form one; the free end of the ligature is tucked into the medial niche and turned just over the arch.

B. Shows the making of a loop attached to a buccal arch:
  A. Attachment of wire for loop.
  B. Formation of loop by gripping the loop-wire just above its attachment with round-nosed pliers and pulling it round one beak.
  C. The wire is now pulled round the arch and finished off as shown in D; the tiny coil round the arch is freely movable on the arch and allows the loop to be made smaller or larger.
  D. The arch is held in the tube by a stainless steel wire ligature.

C. Shows a soft lock-wire for buccal arches in horizontal tubes; the wire passes farther in the niche between tube and band than appears in the illustration.

(From Bennett's 'The Science and Practice of Dental Surgery.'—*Oxford Medical Publications.*)

When a labial bow is employed, the ends pass through horizontal tubes soldered to the anchor bands; the length of the arch in front of the tubes is adjustable by means of loops attached to the arch, one on each side, 5 mm. in front of the tube ; the médial end of the loop is soldered to the arch, the other end of the loop is free and curled round the arch so as to be movable along it ; this end engages the medial aspect of the tube (fig. 188).

The lingual bow may be attached to the bands by the device shown in fig. 189. A half-round post, which fits very accurately a half-round tube soldered on the band in a vertical position, is soldered to the arch. A soft lock-wire is attached to the bow to prevent it being displaced, preferably at both ends in the manner shown for one end. This type of attachment may also be used for buccal arches. A model showing a lingual arch in position is shown in fig. 190. These arches may be used as the active agent in producing the force to move the teeth, or they may serve as a base to which auxiliary springs are soldered and then these latter move the teeth ; the two uses may be combined.

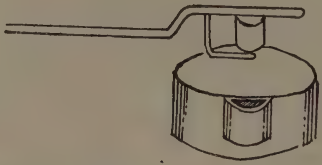


Fig. 189.—Locking device for attaching the lingual arch to the anchor bands.
(By permission of Messrs. H. Kimpton, from 'Applied Orthodontia,' J. D. McCoy.)

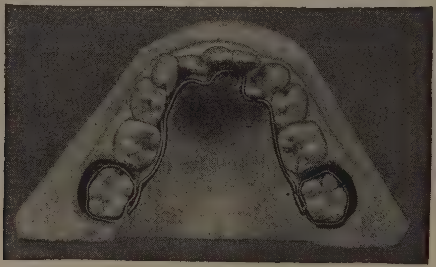


Fig. 190.—Showing lingual arch wire correctly adapted to the mandibular teeth, *i.e.* it is passive in its relation to all the teeth.
(By permission of Messrs. H. Kimpton, from 'Applied Orthodontia,' J. D. McCoy.)

The labial arch is usually used to develop force, though at times auxiliary springs are added to it. The lingual arch is used to produce general lateral expansion, and auxiliary springs, soldered to it, are used to move the six anterior teeth labially (fig. 191) and for the movement of any individual tooth; the lateral expansion is obtained by the gradual opening of a U-shaped loop in the centre of the arch (fig. 192). The lingual arch is fitted to rest at the necks of the teeth, a loop either being made in the arch itself, or one, using wire a gauge smaller than the arch, soldered in it immediately behind the mandibular first incisors; the intervening section of the arch is removed (fig. 193). To expand the arch, not only must the loop be opened but a counter

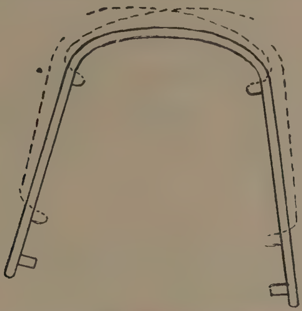


Fig. 191.—Diagram showing convenient locations for the attachment of auxiliary springs.

(By permission of Messrs. H. Kimpton, from 'Applied Orthodontia,' J. D. McCoy.)

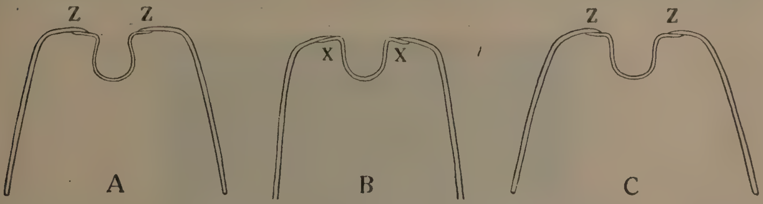


Fig. 192.

A. Lingual arch with soldered-in loop.

B. The same arch with loop opened by forcing one beak of a pair of round-nosed pliers into the loop; the effect is to bring the extremities of the arch closer together.

C. The increased width of the arch is obtained by making counter bends at X, X so as to bring the parts Z, Z to correspond with one another as they did in A. The relationship of the loop to the arch must not be changed, or displacement of anchor teeth would result. The amount of change made to the arch at one alteration should be much less than shown in the diagram.

bend must be made on each side at the point where the U-shaped loop begins. When these changes are made great care must be taken not to change the relationship of the loop to the remainder of the arch. The loops should be opened every eight to ten weeks; 1 to 2 mm. increased breadth in the region of the anchor teeth at any one time is ample.

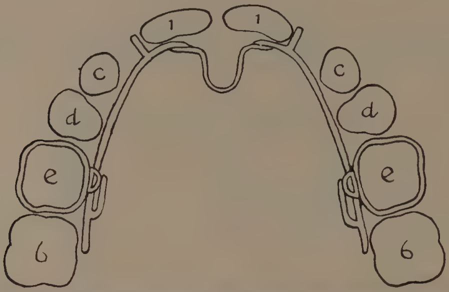


Fig. 193.—Maxillary lingual arch with soldered-in loop and spurs to hold the auxiliary springs *in situ.*

(From Bennett's 'The Science and Practice of Dental Surgery.'—*Oxford Medical Publications.*)

Fig. 194 is shown to emphasise the importance of correctly adjusting the arch to lie accurately in relation to the teeth and tubes on the anchor bands in the first instance; in this case the permanent first molars need buccal movement or expansion, but if the arch were shaped to lie in the mouth, as illustrated, and then fixed to its attachments, the molars would be moved lingually instead of buccally. Slight movement may be obtained

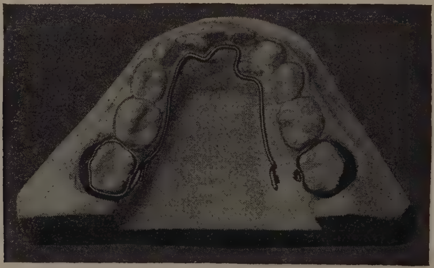


Fig. 194.—Incorrect adaptation of lingual arch wire. If locked in place in this state, the anchor teeth will be subjected to disadvantageous pressure.

(By permission of Messrs. H. Kimpton, from 'Applied Orthodontia,' J. D. McCoy.)

by lengthening the arch itself by stretching with pliers, or by adding solder to it to bear upon the teeth to be moved.

The original type of labial arch was used for all tooth movements, and on account of the comparatively simple technique necessary for its use, a description follows.

It also engages horizontal tubes on the anchor bands, but its ends are threaded and carry a nut whereby its length is adjusted; teeth to be moved labially or buccally are attached to the arch by wire ligatures; lingual movement of the teeth is produced by pressure of the arch upon them. The arch must be carefully fitted in the first instance, and adjusted from time to time, so that the appropriate tooth movements may be accomplished, *i.e.* free of those to be moved labially and touching those to be moved lingually; the arch must not stand so far away from the teeth as to cause discomfort.

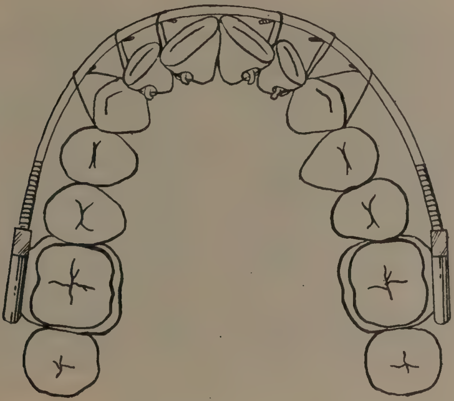


Fig. 195.
(By permission of Messrs. H. Kimpton, from 'Applied Orthodontia,' J. D. McCoy.)

It is usually necessary to fix bands, carrying spurs which will be engaged by a ligature, to teeth which are to be rotated (fig. 195). The threaded end and nut of this arch may well be replaced by a smooth or plain arch to which the adjustable device shown in fig. 188 has been attached.

An auxiliary spring is soldered to the gingival side of the arch at an angle of 45° towards the front of the mouth, to the main wire; the spring is then pulled across the arch at right angles, and again bent back to lie approximately parallel with the arch; it is now given the curvature required (fig. 196).

Convenient situations for the attachment of auxiliary springs are shown in fig. 191.

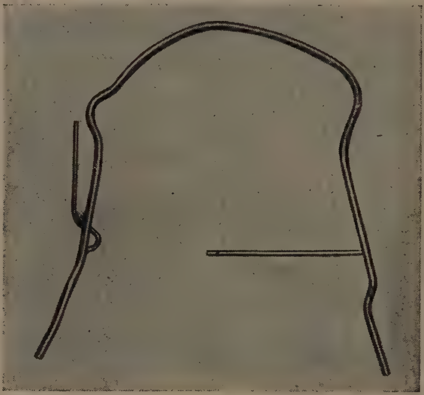


Fig. 196.—Showing method of attaching auxiliary springs to the lingual arch. (J. V. Mershon.) (*Dental Cosmos.*)

In fig. 197 the position of the incisor teeth has been corrected by means of two springs attached to the main arch, one on each side opposite the premolar teeth. Spurs on the arch must be used to keep the springs at the necks of the teeth and to prevent their displacement.

The rotation of teeth may be accomplished by cementing a

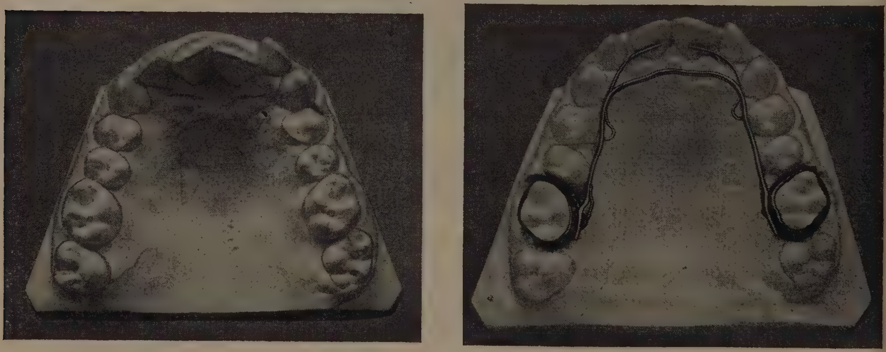


Fig. 197.—To show the use of auxiliary springs attached to a labial arch. (J. V. Mershon.) (*Dental Cosmos.*)

band to the tooth with a spur attached to the labial side of the band (fig. 198). An auxiliary spring is soldered to the arch at a distance from the tooth to be rotated and bent to lie parallel to the main arch. Over the free end of the spring, which has been bent previously to rest some distance lingual to the arch, a fine ligature wire is placed and passed through the gingival space to engage the spur on the band. The ligature wire should then be tightened until the auxiliary spring is brought to lie gingival to the lingual arch. Rotation may also be obtained in the case of an incisor by two springs pressing on opposite surfaces and at opposite corners.

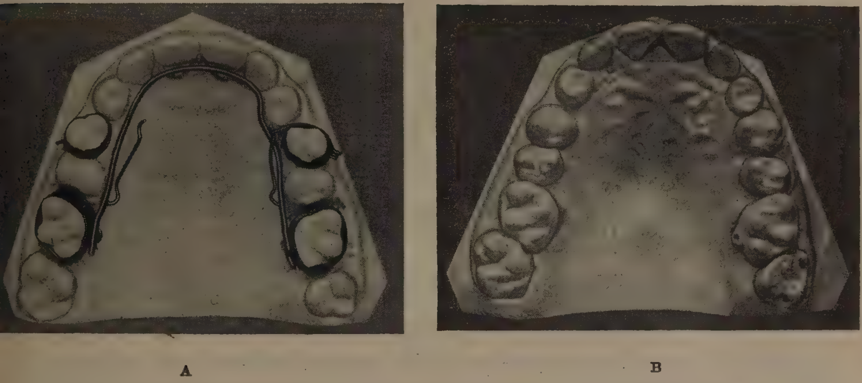


FIG. 198.—Case showing A, springs in position ; B, the teeth rotated. (J. V. Mershon.) (*Dental Cosmos.*)

The inclinational movement (tipping) of the teeth may be achieved by applying pressure as near as possible to the cutting edge ; the movement may be aided by actual pressure or simple resistance from another arch at the cervical edge of the opposite surface of the tooth.

The foregoing examples of mechanical appliances are merely intended to indicate one of the types of fixed appliances in common use for the movement of teeth. The works of Angle, Bennett, Dewey, Lischer, McCoy, and Anderson contain full accounts of the various forms of apparatus.

Choosing between the Use of Removable and Fixed Apparatus.—In deciding upon the particular apparatus to be used, the peculiarities of the case to be treated must be considered. Removable apparatus has an advantage in that both the apparatus and the mouth can easily be kept clean. Moreover, with fixed apparatus the adjustment of the bands to the teeth must necessarily entail some damage to the gingival margin even in the most skilful hands, a point of importance in relation to parodontal disease.

(*c*) **Changes in the Tissues produced by Mechanical Movement.**—The position which a tooth assumes when acted upon by mechanical appliances depends largely upon the manner in which force is applied.

With the majority of appliances a movement similar to that shown in fig. 199 takes place. The force P, which is applied to the crown, is transmitted to the opposing portion of the alveolar process F, and in proportion to the resistance here met with, the apex is moved in the opposite direction. The movement of the apex is probably only slight, and for practical purposes **the tooth may be said to move in the arc of a circle,** the centre of which is represented by the apex of the tooth.

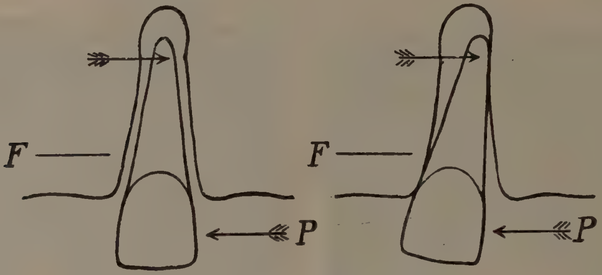


Fig. 199.

When a gentle constant pressure is applied to a tooth, the alveolar wall, against which the pressure is indirectly applied, undergoes absorption, and it has been shown by Gottlieb that, in dogs, there is a compensating deposition of bone on the external aspect of the alveolar wall opposite the point at which the pressure is applied. When the tooth has been moved into the position required and is retained there firmly by suitable means, a fresh deposition of bone takes place and a new socket is formed. The rapidity with which this fresh bone is deposited depends upon (1) the recuperative powers of the patient ; (2) the retention or non-retention of the tooth firmly in its new position by suitable means during the period the tissue is forming ; (3) the amount of local disturbance caused by the operation.

(*d*) Complications and Sequelæ of Mechanical Movement of the Teeth.

(i) **Periodontitis.**—Injury to the periodontal membrane may result from the application of too great a force to the tooth or from direct impingement of the force on the membrane through faulty adjustment. The inflammation of the periodontal membrane may spread to the pulp and so cause death. Chronic periodontitis or even parodontal disease often attacks teeth which have been moved. The changes which occur during orthodontic treatment may tend to lower the vitality of the tissues and so render them liable to attack.

(ii) **Caries.**—This is to a great extent an avoidable sequela. The cleaner the apparatus and mouth are kept the less will be the liability to caries. Movable types of apparatus have in this respect a decided advantage over fixed types. The mouth and all mechanical appliances must be carefully cleaned after every meal. Before inserting the plate an alkaline mouth-wash[1] should be used, and it is also advisable to run a little of the fluid over the surface of the plate where it comes in contact with the teeth.

(iii) **Permanent Enlargement of the Alveoli.**—This condition is due to inadequate re-formation of bone after the tooth has assumed its new position. There may be a lack of recuperative power on the part of the individual from general causes. Growing tissues are more likely to recuperate than tissues which are fully developed, and hence it is that permanent enlargement of the alveoli is frequently met with in cases of regulation in adults. The enlargement of the alveoli may be the result of inflammatory changes in the tissues due to want of care in regulating. Teeth which have been moved should be retained firmly in their new position, otherwise permanent enlargement of the alveoli may result from undue movement in the socket.

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[1] The following is suggested :—

℞. Mag. carb. levis ... ... ʒiv.
Aq. rosæ ... ... ℥vj.
Aquam ... ... ad ℥xij.
M. Shake before using.

After the mouth has been thoroughly cleansed, a tablespoonful is to be taken and moved about in the mouth between the teeth. The magnesia, which is only in suspension, clings about the necks of the teeth and neutralises any acid. Ordinary solutions of alkalies have only a transient effect upon the oral secretions.

(iv) **Injury of the Gums.**—In cases where the regulation has been too rapidly carried out, inflammation of the gums frequently occurs. Inflammation may also arise from want of care in cleansing the plate. The application of a little tincture of iodine, powdered tannic acid, or some other suitable astringent generally removes the trouble. If, however, the inflammation increases, the application of force must be postponed until the gums return to normal condition.

If, owing to defective adjustment, the plate presses on the gums and not on the teeth, sloughing of the soft tissues may occur. Under these conditions the regulating appliance must be immediately abandoned and must not be used again until the tissues have assumed a healthy condition.

(C) THE MOVEMENT OF TEETH BY SURGICAL METHODS.

The immediate regulation of teeth may be carried out by various surgical methods. W. H. Dolamore, who had extensive experience of these, suggested the following as one *modus operandi* :—

The mouth should be freed from sepsis. Using a modified Hey's saw, with a very thin blade, the gum and alveolar process are sawn through midway between the teeth on both sides and nearly to the apex of the misplaced tooth. The tooth is then moved towards its correct position, the movement being completed by forcing on a metal cap splint made to a corrected model. The splint, lined with cement, fixes the moved tooth or teeth in the new position. It is left on for at least six weeks. The cut is carried through the alveolar process merely for convenience. When moving a tooth outwards the inner alveolar plate does not move. A block of gutta-percha or rubber is used between the blades of the forceps to prevent the tooth from jumping out and the blades of the forceps covered with lead foil, lint, or cotton-wool to protect the teeth from damage. A full account of this method will be found in the paper by W. H. Dolamore.[1]

Immediate Torsion.—This method saves time and avoids a plate, but, on the other hand, it involves the following risks :—

(*a*) Liability of the tooth to leave the socket (completely) during the operation.

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[1] *Trans. Odonto. Soc.*, xxxii, 42.

(*b*) The risk of death of the pulp from strangulation of the vessels of the apex.

(*c*) Fracture of portions of the enamel from pressure of the forceps.

(*d*) Fracture of the root in cases where the end is twisted.

As a rule, immediate torsion should not be resorted to after the root of the tooth is complete. Where there is reason to suspect that the root may be twisted, a radiogram should be obtained with the object of ascertaining its shape.

A model of the mouth should be taken before the operation, the tooth corresponding to the one which is to be rotated being cut off and then re-affixed to the model in the position which it will occupy after rotation. A thin metal splint should next be made, by the corrected model, to cover the lower third of the tooth to be operated on as well as the approximal teeth. This splint is applied by first drying the teeth and then fixing the splint in position with osteoplastic cement.

The operation of twisting the tooth should be performed with a pair of forceps which fits the tooth accurately. The blades should be covered with some such substance as thin lead foil, lint, or cotton-wool. W. H. Dolamore recommended the use of india-rubber or gutta-percha between the blades at their division, their elasticity exerting a force in an upward direction. The tooth should be grasped firmly, and slowly rotated, steady pressure in an upward direction being maintained during the process of turning in order to overcome the tendency of the tooth to leave the socket. It is advisable to turn the tooth slightly more than is required. Immediately the operation is completed, the splint should be applied and retained in place for about a week. An antiseptic mouth-wash should be prescribed.

Before the operation is performed it is obviously essential to obtain sufficient space to allow of the necessary movement of the tooth. At times it is difficult to gauge the precise amount of space that will be necessary, and the direction of the root of the tooth to be turned should always be taken into account, as the tooth will not necessarily occupy the whole space.

Should the accident of complete removal of the tooth from its socket occur, the tooth must be cleansed in an antiseptic solution and replaced. Such teeth frequently remain alive.

If the tooth after rotation shows signs of inflammation of the pulp, the gum should be painted with a counter-irritant, but if this fails to bring relief and the symptoms become aggravated the pulp cavity should be opened and the pulp removed. It is inadvisable to delay the removal of the pulp, as timely interference may prevent discoloration of the tooth substance, or, what is more serious, suppuration, which may eventually lead to loss of the tooth.

Instanding maxillary incisor teeth may often be forcibly and immediately moved into place by surgical means; the risks, contra-indications, and after-treatment are very similar to those of immediate torsion. Special forceps (Grever's forceps) having a T-shaped attachment to the outer blade of the forceps shaped to fit the outer alveolar curve are made for this operation.

Owing to risk of accident, surgical methods for moving teeth are not to be recommended when gradual methods may be used.

So far the methods of treatment have only been described in a general way, and it is now necessary to enter into fuller details as to the methods to be adopted in individual cases. For this purpose irregularities may be divided into clinical groups and the following grouping will be adopted :—

(A) Abnormalities in position of individual teeth.

(B) Abnormalities in position of several teeth—General crowding.

(C) Abnormalities of lack of occlusion of many teeth.

(*a*) Superior protrusion.
(*b*) Inferior protrusion—underhung bite.
(*c*) Lack of occlusion—open bite.
(*d*) Abnormalities due to congenital defects of the jaw.

CHAPTER VI

Abnormalities in Position of Individual Teeth.

(A) MAXILLARY INCISORS.

(1) Separation of the First Incisors.

Causes.—Separation of the maxillary first incisors is usually due to the attachment of the frænum of the lip to the mucoperiosteum posterior to the teeth, but occasionally this irregularity can be traced to the presence of a supernumerary tooth; while in rare instances a persistent frænum and a supernumerary tooth are both present. That the frænum acts as a cause of divergent first incisors is often overlooked, but that such is the case will readily be seen on an examination of irregularities of this class. In a few cases the cause is obscure, separation being apparently the result of growth of bone at the median suture. Slight rotation often accompanies separation of the first incisors.

Treatment.—Should the cause of the irregularity be removed before the eruption of the canines, the space between the first incisors will, as a general rule, become lessened without mechanical treatment, as the canines in erupting will exert lateral pressure sufficient to bring the incisors into proper position. Supernumerary teeth should therefore be removed, where necessary, at the earliest opportunity, and the frænum should be cut.

The operation of dividing the frænum is easily performed, as follows: The free edge of the frænum is seized with a pair of artery forceps and drawn forward; a V-shaped piece is then removed from it with a sharp pair of scissors, care being taken to cut the part away where it blends with the gum (a point of great importance). The part between the teeth should also be removed. A strip of lint should be kept between the cut surfaces until the wound has healed, an antiseptic mouth-wash also being given. Cases treated by division of the frænum are shown in figs. 200 to 203. No mechanical treatment was adopted. Division of the frænum, if carried out before the eruption of the canines, will be found to lead to excellent results, but in cases treated after the eruption of these teeth the benefit is not marked, although the space will tend to close slightly. Should the removal of the cause not prove sufficient to correct the deformity, the teeth can easily be approximated by mechanical methods and retained in position awhile.

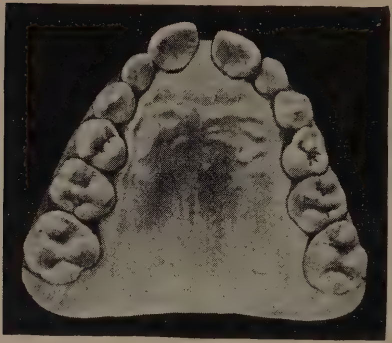

Fig. 200.—Case 1. Before treatment.

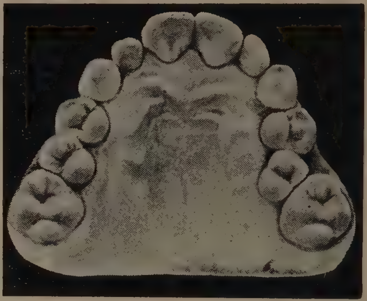

Fig. 201.—Case 1. After treatment.

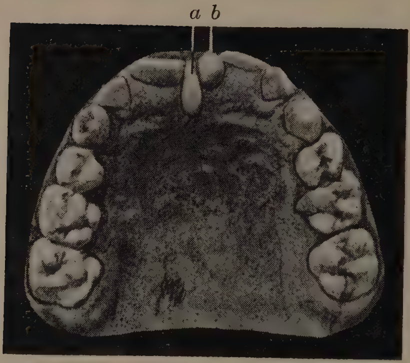

Fig. 202.—Case 2 (semi-diagrammatic). The separation of the first incisors was due to (a) the frænum, and (b) peg-shaped supernumerary tooth.

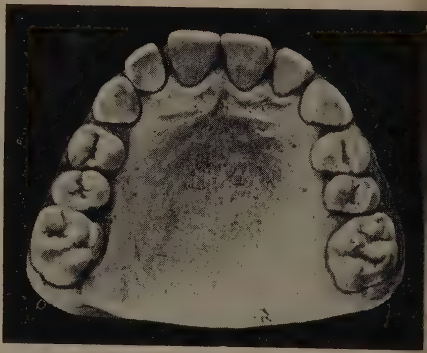

Fig. 203.—Case 2. After treatment.

(2) Displacement External or Internal to the Arch.

Causes.—The displacement of an incisor external or internal to the arch may be due to the persistence of the deciduous predecessor. Where a maxillary incisor erupts so far out of line as to be outside the lower lip, the pressure of the lip will tend greatly to increase the amount of projection. The protrusion of a maxillary incisor may be due to irregularity of the mandibular teeth.

Thumb- or finger-sucking, if continued, will lead to protrusion of the incisors.

In a few instances the protrusion is traceable to the habit of constantly sucking the lower lip or tongue. The prolonged use of the baby comforter or 'dummy' also leads to protrusion of incisors (see pp. 118 *et seq.* and 249). The presence of supernumerary teeth gives rise to cases of displacement which often present considerable difficulties in treatment. An example is shown in fig. 93.

Supernumerary teeth usually develop on the palatal aspect of the arch and cause a displacement of the incisors forwards.

Treatment.—Teeth that erupt internal to the arch may be treated either by an upper plate with wires or wedges (see fig. 180), or by a lower plate with an inclined plane. The latter method is particularly useful where the maxillary teeth do not afford a firm hold for a plate.

When a tooth shows signs of erupting external to the arch it is usually sufficient to remove the deciduous tooth and leave the natural forces to complete the process of correction. But if the tooth falls outside the lower lip mechanical measures must be resorted to. After correction, it will be necessary to retain such teeth in position for from three to six months.

In either case a fixed appliance consisting of a labial arch may be used, the point of delivery of the required force being indirectly the teeth to which the arch is attached.

Where a supernumerary tooth is the disturbing influence, the tooth should be removed as soon as its presence is detected.

(3) Rotation.

Causes.—Rotation of an incisor may arise from the persistence of a deciduous tooth, or from the presence of a supernumerary tooth. A slight tilting of the second incisors may often be traced to the canine developing in a slightly abnormal position. The frænum of the lip, in addition to causing separation of the first incisors, may be the cause of slight rotation of one or both teeth. The amount of rotation of the incisors may vary from an eighth to half a turn.

Treatment.—The first step is to remove the cause, if practicable. In some cases the supernumerary tooth may be high up on the palatal aspect. If the clinical and radiographic appearances indicate that its removal may cause such damage as might involve the misplaced incisor, it is wiser to leave the irregularity uncorrected. The rotation of a tooth may be accomplished either by *immediate torsion*, *i.e.* turning the tooth with forceps, or by *gradual torsion*, *i.e.* rotating the tooth by mechanical methods.

The gradual or mechanical torsion of a tooth can be carried out in a variety of ways, all of which depend upon the principle of two forces working in opposite directions. A favourite method is to fix to the tooth a band with soldered spurs at the mesial and distal angles. Elastic bands are stretched from the spurs to attachments in the vulcanite plate, or ligatures may be used acting from a fixed labial arch and attached to the spurs. Other methods might be enumerated, but they all depend upon employing two forces acting in opposite directions. (See fig. 182.)

(4) Elongation.

Cause.—Elongation of an incisor is usually the result of an accident.

Treatment.—Where the elongation is slight, the tooth may be stoned down by means of carborundum wheels, care being taken to polish the cut surface thoroughly. In more severe cases, an endeavour may be made to force the tooth into the socket if it is desirable to shorten the tooth for æsthetic reasons.

(5) Displacement Upwards of an Incisor.

Causes.—This irregularity is generally the result of injury, but it might result from mal-development.

Treatment.—A radiogram should always be obtained in order to ascertain the shape of the root of the tooth. If the root is normally developed and it is considered advisable to correct the deformity, treatment may be carried out :—

(*a*) By reducing the length of the approximal teeth.

(*b*) By bringing the tooth into place with forceps.

(*c*) By gradually bringing the tooth into place by mechanical appliances.

Method (*a*) may be adopted when the displacement is slight, but it is more suitable for the treatment of the first incisors than for the treatment of the second incisors. With the first incisors a symmetrical appearance can be obtained by stoning down the cutting edge or rounding off the angles of the fellow tooth. With the second incisors reduction of the length of the approximal teeth will naturally give an asymmetrical appearance to the mouth. This may be overcome by treating the incisors and canine on the other side of the mouth in a similar manner ; such treatment is, however, seldom advisable.

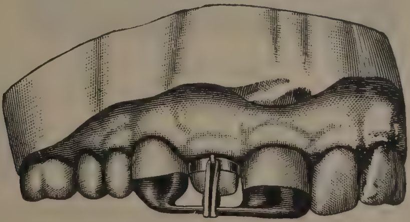

Fig. 204. (From ‘ Text-book of Operative Dentistry ’ (Kirk).)

Method (*b*) may be adopted in cases of forcible displacement upwards, provided that the patient is seen at an early date after the accident.

Method (*c*) may be accomplished by the plan shown in fig. 204, the suggestion of C. L. Goddard. Caps connected by a wire are attached to the teeth approximal to the one to be regulated. To the latter tooth a band is adjusted, hooks being soldered to the labial and lingual surfaces. Traction force is obtained by an elastic band arranged as shown in the illustration.

Where the root of the tooth is abnormal in direction, method (*a*) only should be adopted.

(6) Total Displacement of the Incisors.

Incisors may be transposed, that is, occupy the site of another tooth. For instance, the first and second incisors may change positions, or the second incisor may occupy the position of the canine. Cases of transposition do not call for treatment. The incisors may be completely displaced, as shown in fig. 205, and it is needless to say that the only treatment for such abnormalities is extraction. Occasionally the incisors erupt high up in the alveolar arch and with the cutting edges directed forward. Such teeth are usually 'dilacerated,' and endeavours to bring them into place would prove futile. Extraction is the only remedy.

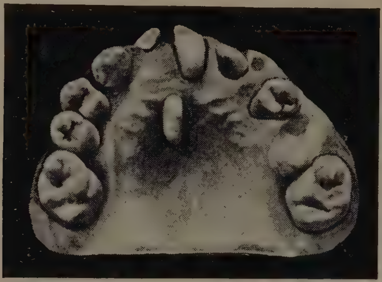

Fig. 205.

Niemeyer [1] records a case in which the maxillary right first incisor erupted through the lip in a woman about sixty years of age; and Salter [2] mentions a case where the second incisor erupted in the nostril.

(B) MANDIBULAR INCISORS.

The mandibular incisors may erupt internal or external to the arch. These conditions are sometimes associated with persistence of deciduous teeth, and when this is the case the obstructing tooth should be removed. No further treatment is as a rule necessary, the pressure of the tongue on the one hand and of the lips on the other usually being sufficient to bring the tooth into line. In a few cases the incisor may occlude outside the maxillary incisor; under these conditions the mandibular tooth must be retracted or the maxillary tooth pushed out. Total displacement of the mandibular incisors is very rare.

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[1] *Deutsche Monatsschrift für Zahnheilkunde.*

[2] 'Dental Pathology and Surgery,' S. J. Salter, 1874, 51.

(C) MAXILLARY CANINES.

The eruption of the maxillary canine external to the arch may be associated with persistence of the deciduous tooth, but as a rule this irregularity is the result of lack of space arising from the forward movement of the premolars and molars. The early extraction of the deciduous second molar may be followed by a forward movement of the permanent first molar, and the second premolar in erupting forces forward the first premolar. If, however, the canine erupts before the second premolar, the first premolar is driven back by the canine and the second premolar erupts in a misplaced position.

Eruption of the canine internal to the arch is usually due to some developmental defect, as pointed out on p. 89.

For the methods of treating the above irregularities, see p. 224.

Partial eruption of the canine occurs occasionally. An effort may be made to force the tooth to erupt, but success is by no means certain. An appliance for this purpose is figured by Essig.[1] A band is cemented to the first premolar. To this band a tube is soldered. Into this tube a piece of wire is fixed which extends across the space and under the cutting edge of the second incisor. A stud is fixed into the canine tooth and traction applied by means of elastic.

In all cases where a permanent canine erupts late, a radiogram should be obtained, and if there is no indication that the tooth is coming into correct position, or the root shows signs of being twisted, the tooth should be removed and the deciduous canine retained.

Complete Misplacement.—The maxillary canine is more frequently 'transposed' than any other tooth, that is, occupies the normal position of another tooth. It may be found usurping the place of the second incisor, or of the first premolar and occasionally of the second premolar. In one recorded case [2] the left canine erupted posterior to the second premolar, the permanent first molar having been extracted at an early age. The canine is totally misplaced from the arch more frequently than any other tooth. The misplaced tooth in these cases often lies dormant and causes no inconvenience, but, should it erupt, extraction is the best remedy. A unique case in which a deciduous canine was developing within the left orbit has been recorded by J. W. Cousins.[1] The patient was two years of age and there was a hard swelling located just within the left orbit. On the growth being detached from its surroundings, the crown of a deciduous canine was found enclosed in a sac, the root of the tooth being attached to the orbital plate by fibro-cartilage.

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[1] 'Text-book of Prosthetic Dentistry.'

[2] Hopewell-Smith, *Brit. Dent. Journ.*, April 15, 1914.

Misplaced canines may lead to troublesome irregularities of the teeth present in the arch. An example is shown in fig. 206.

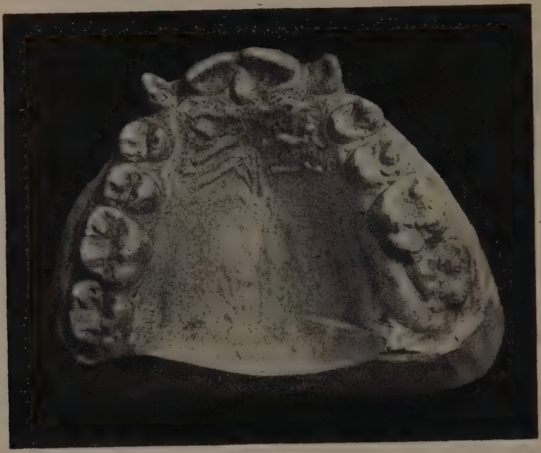

FIG. 206.

The second incisors have been rotated and tilted owing to the pressure of the canines. Under such conditions, the canines should usually be removed and correction of the misplaced teeth carried out.

(D) MANDIBULAR CANINES.

Irregularities of the mandibular canines from causes other than crowding are rare. In the case shown in fig. 107 the deciduous canine is in position and the permanent canine has erupted internal to the arch. The misplaced tooth was removed.

An interesting example of a displaced mandibular canine was recorded by C. Truman.[2] The tooth appeared under the chin, the presence of a sinus having apparently caused the deviation. After its removal, a premolar made its appearance in the same position. A similar case was recorded by D. Whittles.[1]

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[1] *Brit. Dent. Journ.*, viii, 294.

[2] *Trans. Odonto. Soc.*, xxiii, New Series, 34.

Complete misplacement of the mandibular canine has been referred to on p. 96.

(E) PREMOLARS.

Abnormalities in the position of the maxillary and mandibular premolars are usually the result of early extraction of the deciduous second molar. The first premolar erupts in correct position with the second premolar displaced internal to the arch. More rarely, both the teeth are involved in the irregularity, as seen in fig. 207. Irregularly placed premolars are best treated by extraction, provided that both the approximal teeth can be saved. If, however, one of the approximal teeth is unsavable it should be removed, and the displaced premolar moved into position. In a case similar to that shown in fig. 207, one of the premolars should be removed, and, in choosing between them, the judgment should be guided by their relation to the opposing teeth and by the degree of rotation.

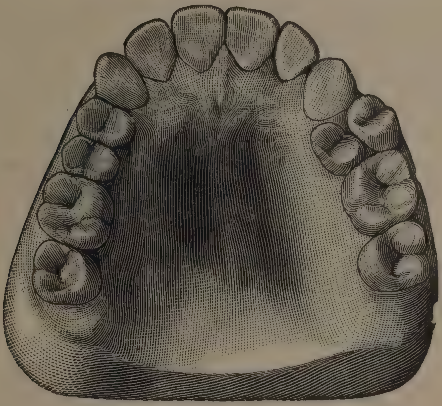

Fig. 207.

Displacement of the premolar may be associated with persistence of the deciduous second molar. The probable cause of this irregularity was discussed on p. 93.

Occasionally the premolars are completely rotated, the external cusp being placed towards the palate. This irregularity is at times symmetrical.

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[1] *Brit. Dent. Journ.*, December 1901.

Transposition of the maxillary canine and first premolar may occur. Complete displacement of the premolars from the arch is rare.

(F) MOLARS.

The irregularities in position of these teeth have already been described on pp. 94 and 99. When they affect the first or second molars they may, if taken early, be moved into correct occlusion by either removable or fixed appliances, though this is not always by any means easy, but misplaced or non-functional third molars should be removed as they are always potential sources of harm.

Note.—As the displacement of permanent teeth is so often due to natural forces acting after the too early loss of deciduous teeth from disease, and particularly from the movement or tilting forwards of permanent first molars after the loss of deciduous molars, it is good treatment when the deciduous molars must be lost early to insert an appliance to prevent this forward movement ; but care must be taken that the normal eruption of teeth is not interfered with or injury to the gingival margin brought about by lack of cleanliness.

CHAPTER VII

Abnormalities in Position of Several Teeth—General Crowding.

In cases of general crowding of the teeth the irregularity varies from a slightly crowded arrangement of all the teeth to the extrusion from the arch of one or several teeth. The general causes of abnormalities in position of the teeth have been discussed in Chapter IV ; here it may be stated that crowding of the teeth is to be traced to a reduction in the growth of the jaw due to inherent or acquired causes, with no concomitant reduction in the size of the teeth, and to the self-adjusting movements of the teeth where some of them have been prematurely lost.

(A) MORBID ANATOMY AND PATHOLOGY.

The positions which teeth assume in the crowded arch depend upon several contingencies which may now be conveniently referred to. The direction in which a tooth erupts is in all probability identical with the line of the long axis of the tooth in the crypt, and that being so, it follows that the position of a tooth in its crypt is an important factor in determining the position which it will assume in the arch. Normally, the developing permanent teeth in the maxilla are arranged as follows : The crown of the first incisor slopes slightly forwards ; the second incisor lies in a plane posterior to that of the first incisor, the distal border being in close proximity to the anterior root of the deciduous first molar ; the permanent canine is high up in the bone and lies in a plane anterior to that of the second incisor ; the premolars are embraced by the roots of the deciduous molars (fig. 208). In the mandible a somewhat similar arrangement exists. With a diminution of space available for the teeth, it necessarily follows that any irregularity in position of the teeth in their crypts becomes more marked. The relative positions as seen in the normal may remain, the second incisors being placed more inwards and nearer to the middle line with the canines more

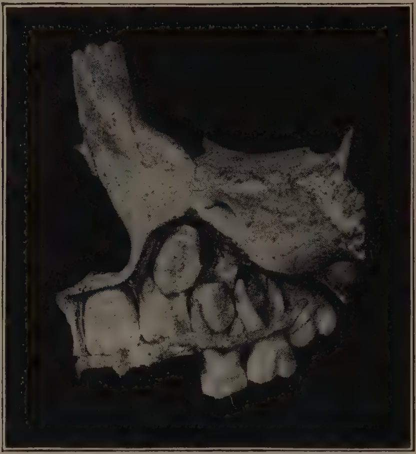


FIG. 208.—Left maxilla of a child six years of age, to show the position of the permanent teeth.

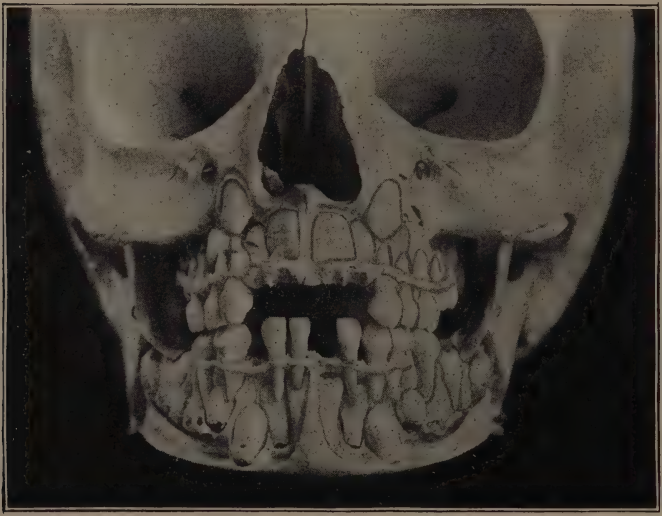


FIG. 209.—Skull and mandible of a child aged five years six months, showing irregularity in position of the maxillary right incisors.

outwards, or the *normal relative positions may be disturbed*, as shown in the following specimens :—

The skull of a child of five years six months is shown in fig. 209. Several of the deciduous teeth have been lost, but those that remain are slightly crowded. In the left maxilla the positions of the first and second incisors in relation to one another are normal ; the first incisor, however, is a little tilted on its long axis. In the right maxilla the first incisor is tilted so that the medial angle is forward, and the second incisor overlaps the distal corner of the labial surface. The sutures are closed in their distal halves.

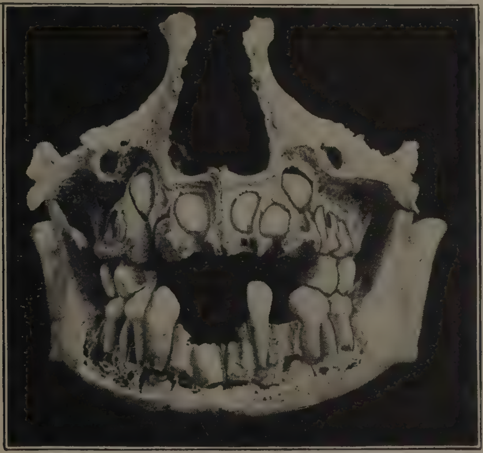


Fig. 210.—In this specimen there is marked irregularity in the position of the developing maxillary incisors.

Fig. 210 reveals a considerable departure from the normal in the left maxilla. The first incisor is rotated so that the medial angle looks almost directly forwards ; the second incisor is also rotated, but to a lesser extent, and overlaps the first incisor. In the right maxilla the first incisor is rotated, the distal angle being directed forwards ; the second incisor is overlapped by the first incisor.

In the next specimen (fig. 211), five years nine months, the maxillary deciduous incisors have been removed to show the position of the permanent teeth. The maxillary first incisors have their medial borders rotated a little forward, and the second incisors overlap their distal corners. The deciduous teeth, when in position, are bent a little inwards, and there is slight crowding. The sutures are partially filled up in their outer quarters, the right a little more than the left. In connection with the closure of the sutures it is interesting to note that the irregularity of the permanent teeth is more marked on the right side.

In fig. 212 the maxillary right second incisor slightly overlaps the first incisor and the medial angle is tilted forward. On the left side the second incisor slightly overlies the first, the latter tooth being a little rotated. The maxillary deciduous incisors are missing, but the mandibular teeth show good spacing. The inter-canine width is slightly above and the inter-molar width equal to the normal average. The suture on the left side runs near to the deciduous canine socket ; the right suture terminates forward of the deciduous canine.

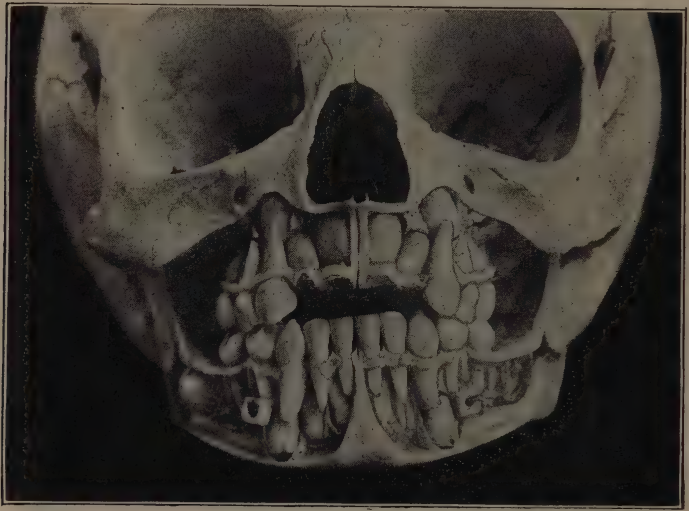


FIG. 211.—Skull and mandible of a child aged five years nine months, with the deciduous incisors removed to show the irregularity in position of the developing maxillary incisors.

Another modification in the position of the permanent incisors is shown in fig. 213. The maxillary first incisors are placed with their long axes towards the mid-line and the canines lie with the crowns pointing away from the mid-line. The opening of the anterior nares is small, and the whole specimen conveys the impression that there is a want of growth of the body of the maxilla.

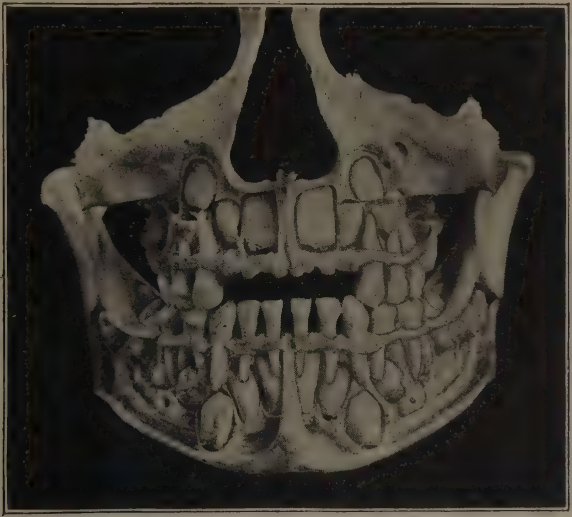


Fig. 212.—Skull and mandible of a child about six years of age, showing irregularity in position of the developing maxillary incisors.

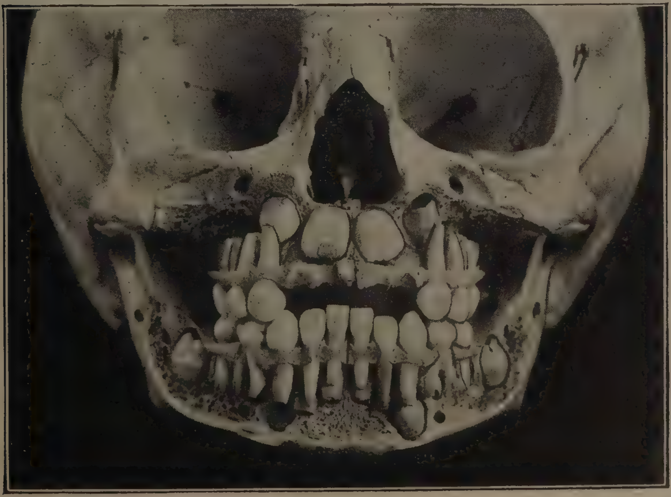


Fig. 213.—Skull and mandible of a child about five years of age, showing considerable irregularity in the position of the developing maxillary incisors and canines.

Fig. 214 is from the skull of a child between six and seven years of age. The maxillary incisors are distinctly crowded; the right first incisor is partially rotated and is overlapped by the second incisor; the left first incisor has a distinct slope away from the mid-line and overlaps the second incisor. The arch of the deciduous teeth is well formed, the teeth are well spaced; the maxillary-premaxillary sutures are closed in their distal halves.

The Order of Eruption is an important factor in determining the character of an irregularity, and this is clearly shown in cases of early loss of the deciduous second molar. When the teeth appear in their normal order the canine erupts after the second premolar; hence, if there is insufficient room in the arch, the canine will be the principal disturbing factor. If there is a fair interval between the second incisor and the first premolar the canine will force its way into the arch, causing disturbance in the position of the incisors, but if the interval is slight the result will be the exclusion of the canine which will erupt external to the arch. The pressure of the canine, *even when excluded from the arch*, may force the incisors into irregular positions.

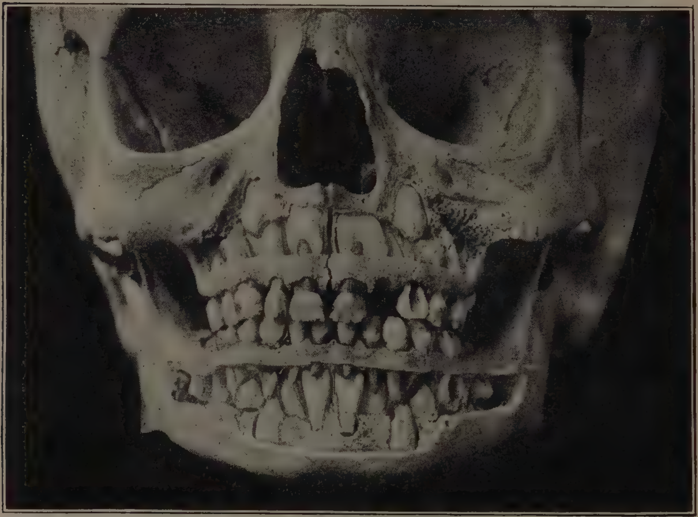


FIG. 214.—Skull and mandible of a child between six and seven years of age. The maxillary incisors are developing in abnormal positions.

Should the canine erupt before the second premolar, the disturbance in the position of the anterior teeth will be less noticeable. The incisors, canine, and first premolar will erupt in a fairly regular manner, the premolar being forced towards the permanent first molar. If the space between the premolar and permanent first molar is slight, the second premolar will erupt internal to the arch and cause little disturbance. On the other hand, if the interval is nearly normal, the second premolar in erupting will, in all probability, cause displacement inwards of the first premolar and so produce a type of saddle-shaped arch.

The type of irregularity also varies with *the cause interfering with the growth of the jaws*. In cases where adenoids have been present before the age of six the interference with the growth of the premaxillæ will lead to crowding of the first and second incisors, and the roots of the second incisors will lie with their apices directed towards the median line. The canine also, owing to the want of development of the anterior nares, will lie too near to the median line, with the result that, when erupted, the root will cover a small portion of the upper part of the second incisor root. This point is of practical importance when the treatment of crowded incisors is under consideration. Where the main interference in the growth has occurred in the molar region, the incisors will generally erupt in a regular curve, and the principal irregularity will be posterior to the premolars and canine, the irregularity being shown in an increased backward direction of the occlusal surfaces of the molars.

In order that the conditions which exist in cases of crowded arches may be fully appreciated, it is essential that a careful study should be made of morbid anatomy specimens. Unfortunately, our dental museums possess few such specimens, but there is a useful collection in the Museum of the Royal College of Surgeons of England, and specimens in this collection will be described.

The maxillæ shown in fig. 215 are from a male aged eighteen years. The teeth are arranged in an arch of the shape of the normal English type. The width across the first premolars is 26 mm.; between the canines 22·3 mm. The incisors are slightly crowded; the right teeth are rotated, the first incisor overlapping its fellow of the opposite side. The maxillary-premaxillary suture runs on the right side to the medial aspect of the canine and on the left side to the distal aspect of the canine. This is instructive, as the greater degree of crowding is on the right side.

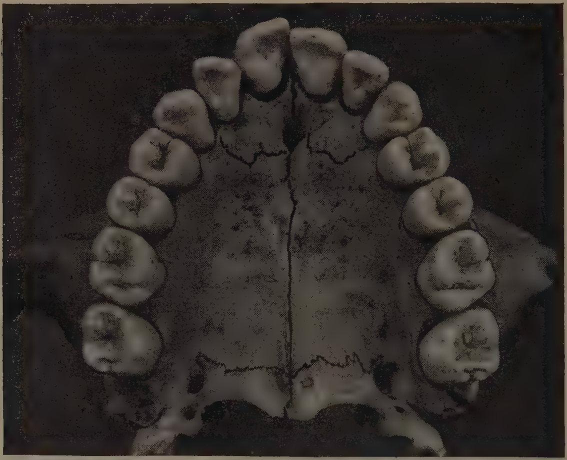


Fig. 215.—Maxillæ showing irregular arrangement of the incisors. (R.C.S. Odonto. Series.)

The positions of the sutures in this and the following specimens are indicated by dark lines.

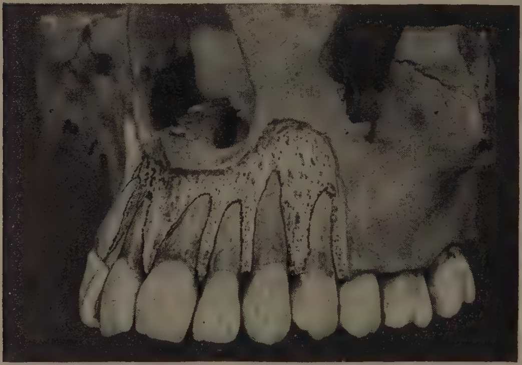


Fig. 216.—Side view (left) of the specimen shown in fig. 215.

A view of the left incisors and canine is shown in fig. 216. The crowns of these teeth slope slightly forwards, the medial angle of the first incisor being beyond the mid-line.

The right side (fig. 217) presents the following instructive features :—

(1) The canine has the crown placed in a slightly backward direction.

(2) There is marked rotation of the second incisor, the medial border looking inwards.

(3) The crown of the first incisor slopes towards the opposite side, the medial angle overlapping the left first incisor.

(4) The root of the second incisor is overlapped by that of the first incisor.

These facts indicate a lack of growth of the premaxillæ.

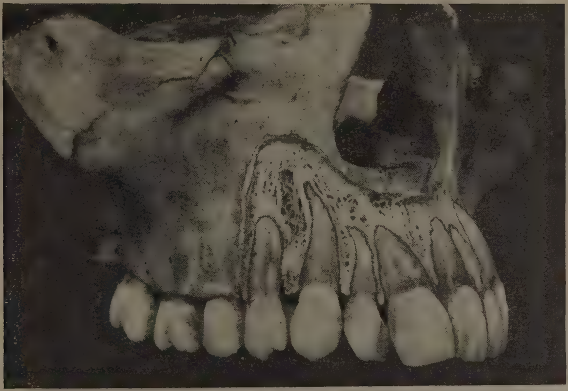


FIG. 217.—Side view (right) of the specimen shown in fig. 215.

An extreme degree of crowding in the incisor region is seen in the specimen illustrated in fig. 218. The width of the arch is :—

Between the first premolars, 25·2 mm.
Between the canines, 20·5 mm.

On the right side, the crowns of the canine and the incisors incline towards the mid-line ; the medial angle of the second incisor is covered by the distal angle of the first incisor, while the latter tooth has the medial angle well across the mid-line. On the left side, the crown of the canine has a marked forward inclination ; the first incisor is rotated so that the medial border looks directly forwards ; the second incisor is placed internal to the arch and is situated immediately posterior to the root of the first incisor. Here again the position of the maxillary-premaxillary suture is instructive. On the right side it runs towards the medial aspect of the canine; on the left side it appears to run directly up to the medial aspect of the second incisor.

The other points to be noted about the specimen are :—

(1) The regular arrangement of the premolars and the first and second molars.

(2) The abnormal shape of the second molars, these teeth appearing to be compressed from before backwards.

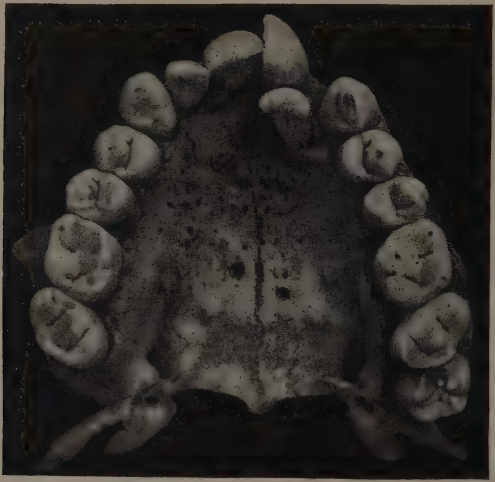


Fig. 218.—A specimen showing marked irregularity in position of the left incisors.

(R.C.S. Odonto. Series.)

(3) The left third molar has erupted high up in the tuberosity, the occluding surface looking well backwards.

(4) The right third molar is congenitally absent.

The irregularity of the incisors is probably due to under-development of the premaxilla, and the malposition of the third molar to insufficient backward growth of the maxilla, a condition often associated with adenoids.

An example of crowding of the anterior teeth in a child aged seven years three months is shown in figs. 219 to 222.

A profile view of the specimen shows that the maxillary first incisors have partly erupted and that the left tooth is inclined inwards. The maxillary second incisors are situated well posterior to the first incisors, the canines overlapping their anterior aspects; this is particularly noticeable on the left side.

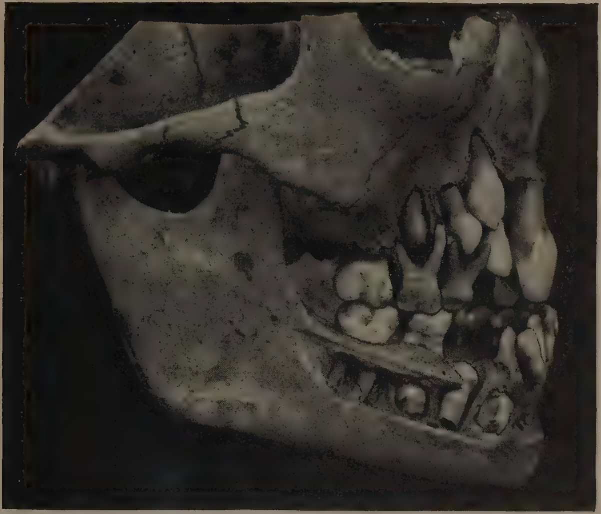


FIG. 219.—Skull and mandible showing crowding of the incisor teeth. (Right side.)

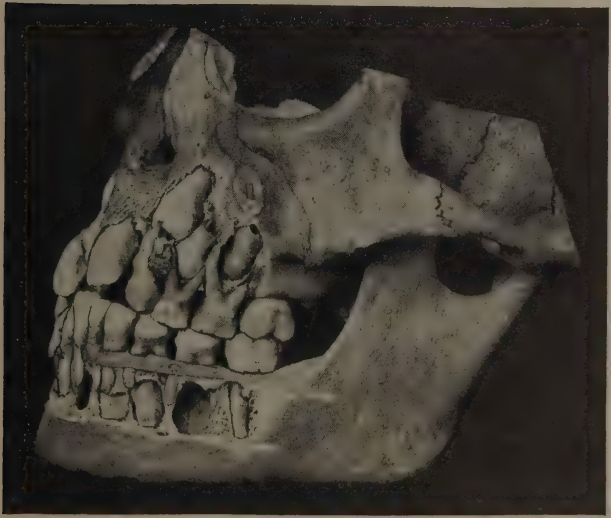


FIG. 220.—Skull and mandible showing crowding of the incisor teeth. (Left side.)

The palatal view (fig. 221) shows that the premaxillæ are very poorly developed and that the anterior part of the palate presents the pushed-up appearance so characteristic of the adenoid palate. The permanent first molars have assumed a normal position in the arch and the second molars appear to have ample room for development.

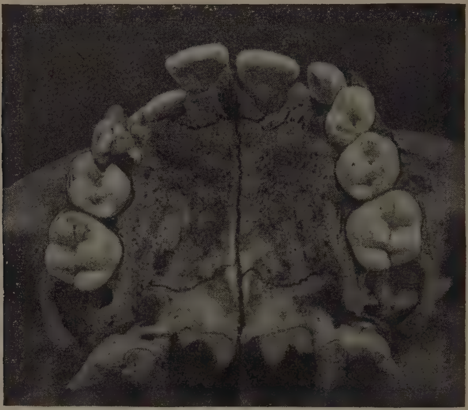


Fig. 221.—Palatal view of the specimen shown in figs. 219 and 220.

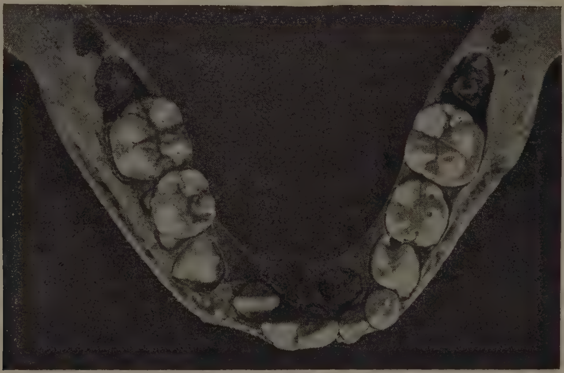


Fig. 222.—View of the mandibular teeth of the specimen shown in figs. 219 and 220.

In the mandible, fig. 222, there is a crowded arrangement of the incisors, the right second incisor lying posterior to the first incisor.

Side views of the jaw are illustrated in figs. 219 and 220. On the left side one of the premolars has unfortunately been lost; on the right side, however, they are all present, so that a good idea can be obtained of the general crowding of the maxillary teeth. The occlusion of the permanent first molars is normal, and there seems to be ample room for the growing premolars. The crown of the first premolar is in close proximity to the second incisor and the anterior aspect is in contact with the crown of the canine. The crowding in this specimen seems to be due to lack of growth of the premaxillæ.

The specimen from a female child aged seven years eight months, shown in figs. 223 to 226, is a good example of marked crowding of the teeth from undergrowth of the maxillæ.

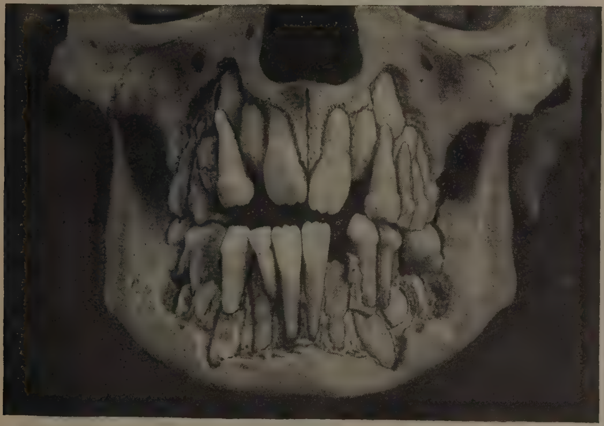


Fig. 223.—Specimen showing general crowding of the teeth. (R.C.S. Odonto. Series.)

The palate (fig. 224) is distinctly small, and towards the front shows the curious pushed-up appearance so often seen in those who have suffered from adenoids in the early years of life. There is also marked asymmetry of the dental arch, the right side diverging more than the left. A view of the left premolars and molars is shown in fig. 225.

The features to be noted are :—

(1) The normal occlusion of the deciduous first molars.

(2) The slight forward movement of the mandibular permanent first molar owing to early loss of the deciduous second molar.

(3) The extreme backward direction of the occlusal aspect of the maxillary permanent first molar.

(4) The crowded arrangement of the developing maxillary premolars. The anterior buccal root of the permanent first molar is almost in contact with the second premolar.

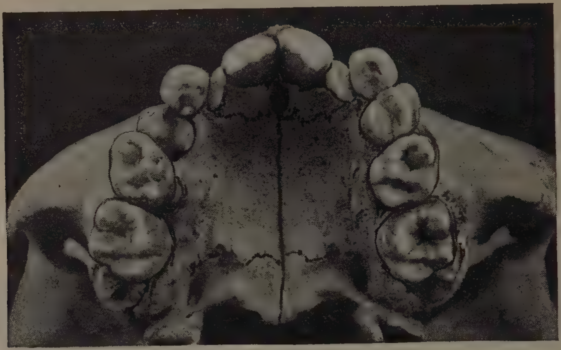


Fig. 224.—Palatal view of the maxillary teeth of the specimen shown in fig. 223.

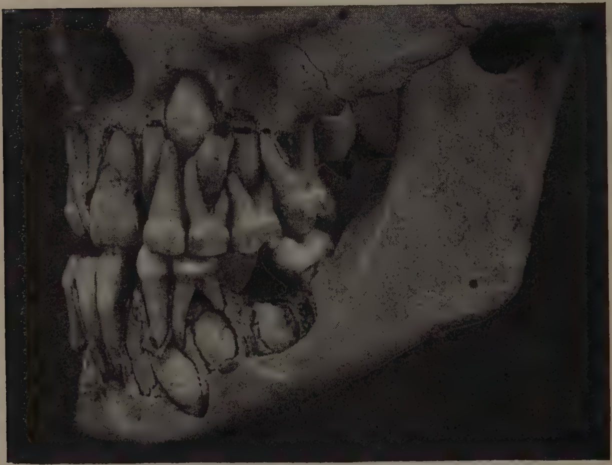


Fig. 225.—Side view (left) of the specimen shown in fig. 223.

(5) The definite inward direction of the second premolar.

(6) In the mandible the apparently ample space for the developing premolars.

On the right side (fig. 226), with the loss of the maxillary deciduous first molar, the deciduous second and permanent first molars have moved forward, placing the maxillary permanent molar in prenormal occlusion to the lower, which though really in normal position now occludes postnormally. The forward movement of the permanent first molar has allowed this tooth to assume a direction in the arch more closely approximating the normal, but the anterior buccal root is still too close to the second premolar. Want of room has caused the second premolar to assume an inward direction. In the mandible there appears to be ample space for the developing teeth.

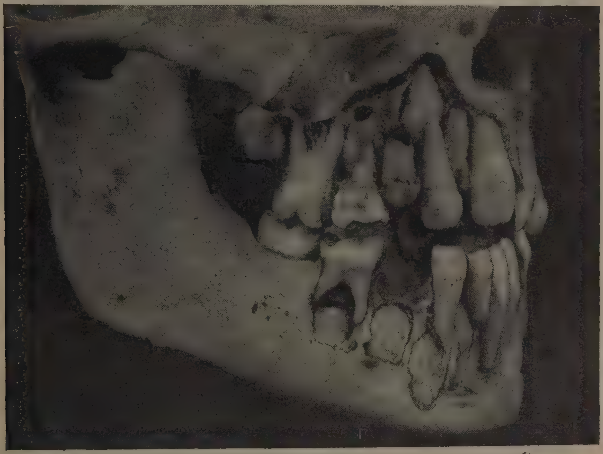


FIG. 226.—Side view (right) of the specimen shown in fig. 223.

The view of the anterior aspect of the specimen (fig. 223) shows that the maxillary incisors are erupting with a distinct inward inclination of the crowns. The teeth are arranged in a crowded manner shown by the rotation and overlapping of the teeth. On the left side (fig. 225) the canine is high up in the bone between the second incisor and the first premolar, the canine having rotated in order to adapt itself to the diminished space. On the right side (fig. 226) the rotation of the maxillary canine is more marked.

An example of general irregularity of the anterior teeth is shown in the skull and mandible illustrated in figs. 227 to 231. The age of the child was eight years six months.

On the left side (fig. 228) the occlusion of the permanent first molars is normal; the maxillary first incisor has erupted internal

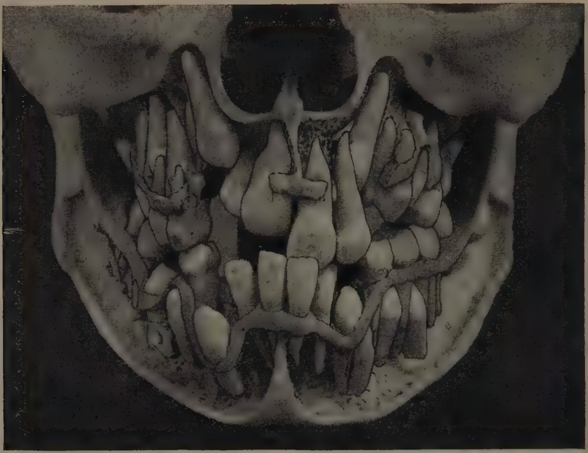


Fig. 227.—Skull and mandible of a child eight years six months, showing general crowding of the anterior teeth.
(R.C.S. Odonto. Series.)

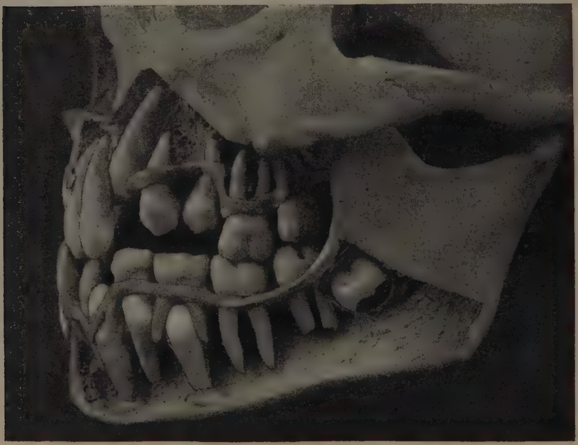


Fig. 228.—View of the left premolars and molars of the specimen shown in fig. 227.

to the bite of the mandibular tooth, which in its turn has been driven slightly forward; the maxillary second incisor has the crown tilted backwards; the crown of the canine has a marked slant towards the mid-line and is in contact with the posterior aspect of the root of the second incisor; the maxillary premolars are erupting with a distinct inward inclination; the mandibular canine and premolars are normal in position.

On the right side (fig. 229), in the maxilla, the first incisor is not fully erupted and is slightly rotated, the medial angle looking backwards; the second incisor is erupting behind the first incisor; the root is directed backwards and inwards, so that a marked space exists between it and the root of the first incisor; the crown of the canine is situated anterior to the second incisor and is erupting with a definite slope towards the median line; the second premolar has the root directed backwards, the distal aspect lying in contact with the anterior buccal root of the permanent first molar.

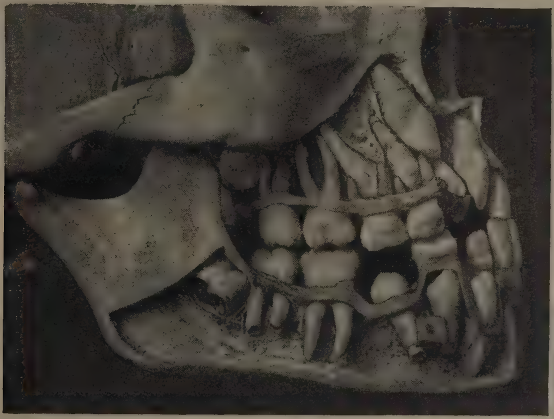


Fig. 229.—View of the right premolars and molars of the specimen shown in figs. 227 and 228.

In the mandible, the canine is displaced outwards; the first premolar is slightly rotated and the crown has an outward inclination; the second premolar has the crown looking backwards and there are spaces between it and the approximal teeth. The permanent first molar does not belong to the specimen.

A palatal view of the maxilla (fig. 230) shows that the premaxillæ are quite small, with the suture between them and the maxillæ still open. The anterior nares are well developed.

The irregularity in this specimen would appear to be due to deficient growth of the premaxillæ.

The specimen shown in fig. 232 is a good example of a crowded arch. The incisors are crowded; there is marked contraction across the region of the first premolars and canines; the second and third molars are misplaced.

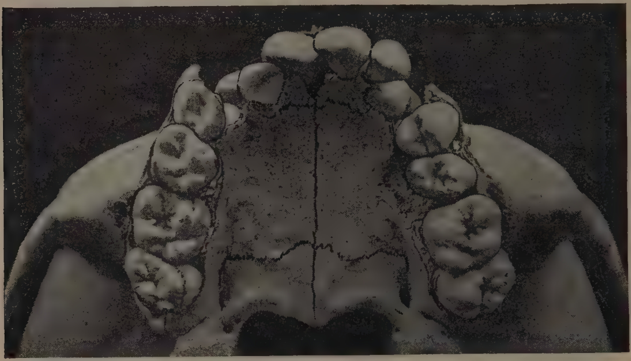


Fig. 230.—Palatal view of the maxillary teeth of the specimen shown in figs. 227 and 228.

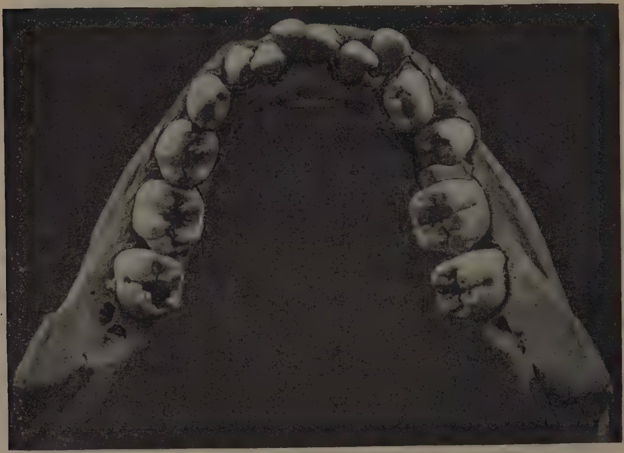


Fig. 231.—View of the mandibular teeth of the specimen shown in figs. 227 and 228.

Viewed from the front (fig. 233) the maxillæ show a pinched appearance; the malar process bends downwards at the junction

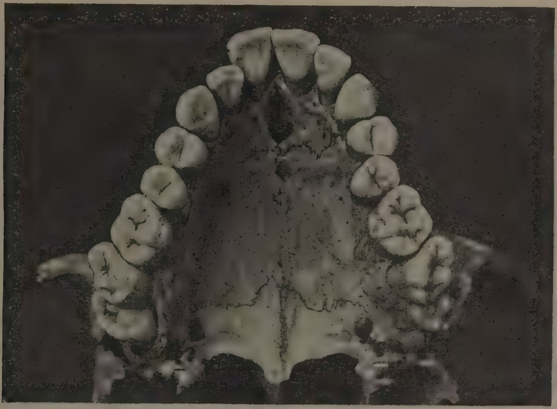


FIG. 232.—Palatal view of a crowded arch in an adult.

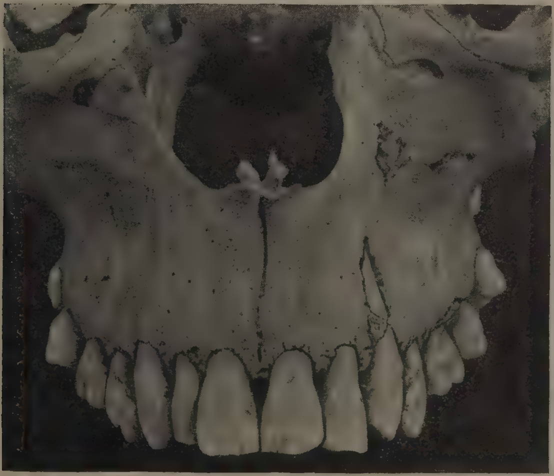


FIG. 233.—Anterior view of the specimen shown in fig. 232.

with the malar bone. Contrast this specimen with that shown in fig. 234, where the teeth are practically normal in position. The facial surface of the bone is well developed and the malar process takes a bold upward sweep. The capacities of the sinuses in the abnormal specimen are—right 90 and left 130 cubic millimetres ; those of the normal specimen, although of an individual only sixteen years of age, are 245 and 205 cubic millimetres.

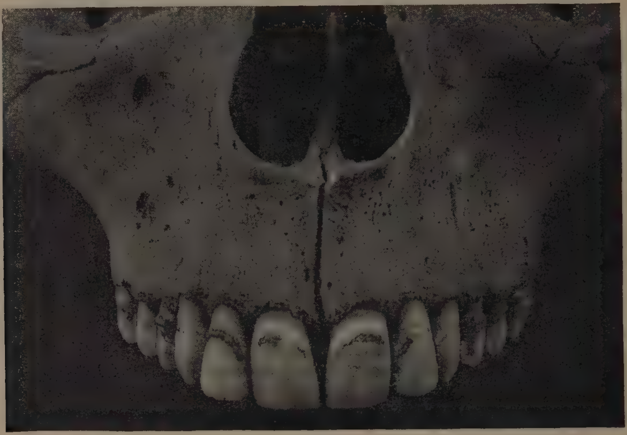


Fig. 234.—Normal maxillæ.

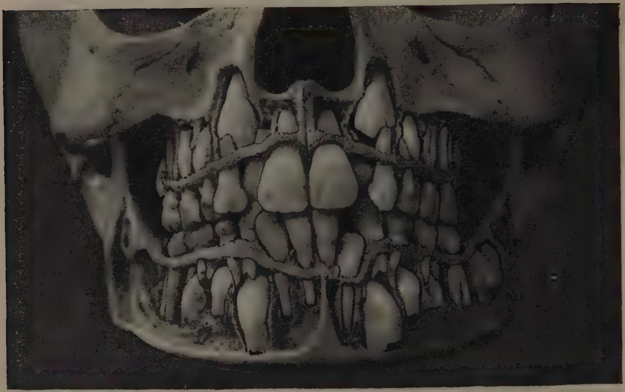


Fig. 235.—Skull and mandible of a child showing crowding of the anterior teeth.

The skull with mandible of a child about seven to eight years of age shown in fig. 235 is a good example of crowding of the incisor teeth associated with an abnormal occlusion of the premolars and molars, and is typical of many cases seen in practice. A palatal view of the maxilla is shown in fig. 236.

The points to be noted are :—

(1) The narrowing of the arch across the region of the first deciduous molars.

(2) The crowded arrangement of the incisors, the second incisors being placed internal to the arch.

(3) The small size of the premaxillæ.

(4) The presence of a suture in each premaxilla, suggesting that the bone has arisen from two centres. The sutures between the premaxillæ and maxillæ skirt the distal aspect of the sockets of the second incisors, and, towards the outer ends, are distinctly filled up, on the left side the suture being completely obliterated in one place. The sutures in the premaxillæ run towards the medial aspects of the second incisors.

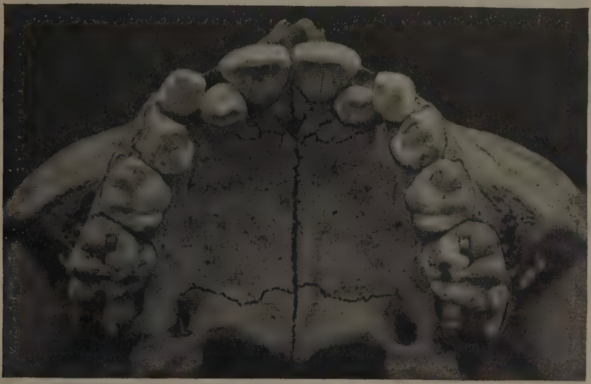


FIG. 236.—Palatal view of the specimen shown in fig. 235.

The occlusion of the mandibular deciduous and permanent molars is postnormal (figs. 237 and 238). The maxillary permanent first molar appears to be in a normal position, the occluding surface looking downwards and slightly backwards and outwards. The premolars are in normal positions.

The positions of the anterior teeth are shown in fig. 235. In the maxillæ, the deciduous canines are less than 2 mm. from the first incisors ; the second incisors lie posterior to the first incisors, the roots being directed towards the mid-line ; the crowns of the permanent canines slope slightly towards the mid-line, the left tooth lying in front of the root of the second incisor and the right tooth lying in contact with the distal aspect of the incisor.

In the mandible, the incisors are crowded; each second incisor is rotated, with the mesial half of the crown posterior to the first incisor; the permanent left canine is almost vertical, while the right tooth has the crown directed away from the median line instead of towards it.

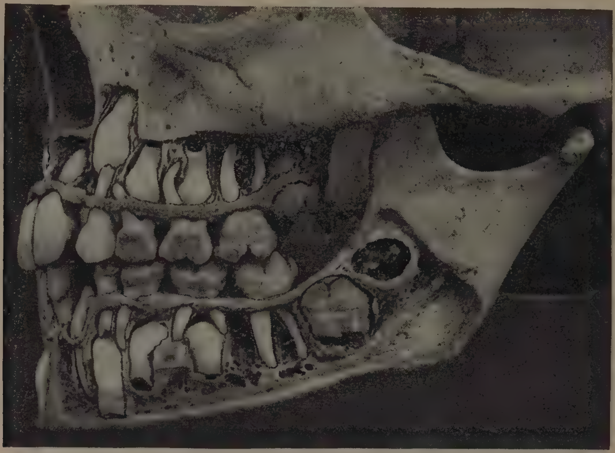


FIG. 237.—View of the left side of the specimen shown in fig. 235.

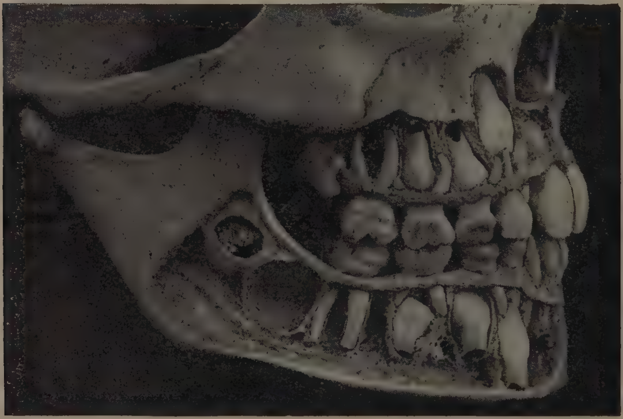


FIG. 238.—View of the right side of the specimen shown in fig. 235.

The irregularity of the teeth in this specimen would appear to be due to a lack of growth on the premaxilla leading to a narrowing of the anterior part of the arch and crowding. The crowding in the mandible is probably secondary to the small maxilla, the latter impeding the forward movement of the mandible.

Fig. 239 shows an example of general crowding of the anterior teeth. The arch of the maxillary teeth is distinctly narrow and the incisor teeth are crowded, the left canine being slightly extruded. The right second premolar and the third molars are missing.

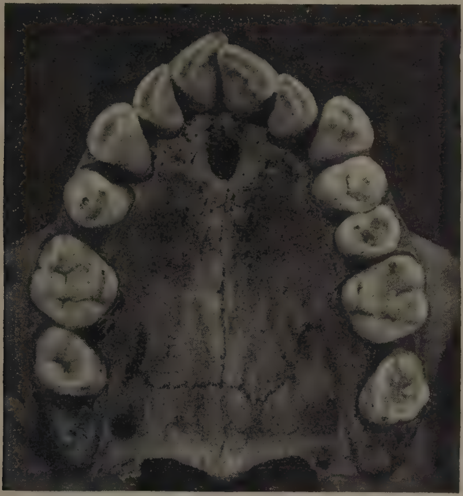


Fig. 239.

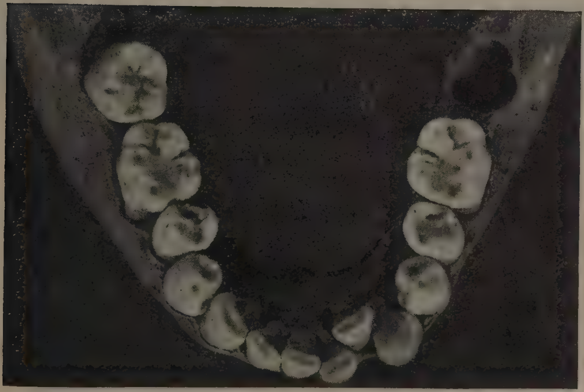


Fig. 240.

In the mandible (fig. 240) the narrowing of the arch has led to the forcing inwards of the left second incisor and slight crowding of the remaining incisors and right first premolar. The third molars are missing.

The occlusion of the teeth is shown in figs. 241 and 242.

On the left side (fig. 241) :—

(*a*) The outer cusps of the maxillary premolars and molars are but slightly external to the outer cusps of the mandibular teeth.

(*b*) The crown of the mandibular canine slopes towards the median line and the cutting edge overlaps the distal angle of the maxillary second incisor.

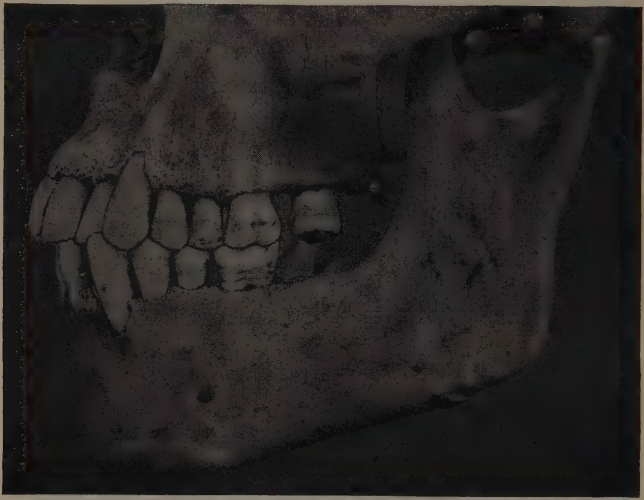


FIG. 241.—View showing the occlusion of the left premolars and molars of the specimen shown in figs. 239 and 240.

(*c*) The crown of the maxillary canine is directed towards the middle line.

(*d*) The mandibular first incisor strikes against the distal angle of the maxillary first incisor.

On the right side (fig. 242) :—

(*a*) The maxillary first premolar and permanent first molar are in flush occlusion with the mandibular teeth.

(*b*) The maxillary second molar occludes internal to the mandibular teeth.

(*c*) The crown of the maxillary canine inclines forwards, the

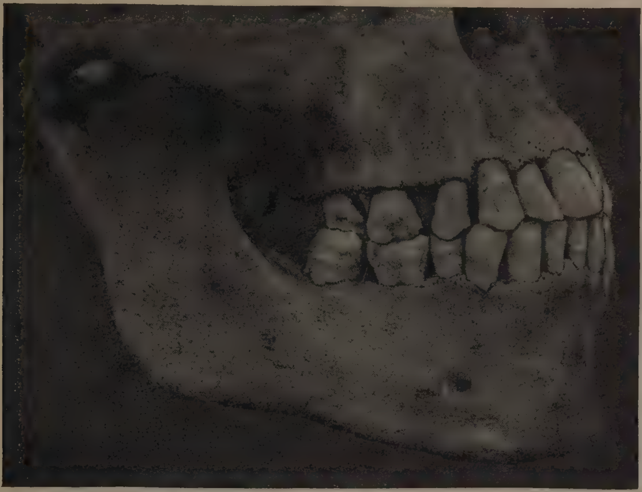


FIG. 242.—View showing the occlusion of the right premolars and molars of the specimen shown in figs. 239 and 240.

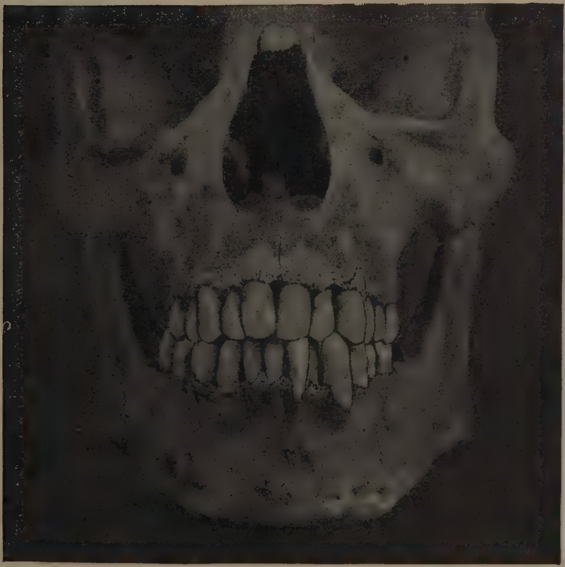


FIG. 243.—Anterior view of the specimen shown in figs. 239 and 240.

distal part of the occlusal margin meeting the anterior part of the mandibular tooth edge to edge.

(*d*) The crowns of the maxillary incisors slope forwards, the first incisor overlapping the tooth on the opposite side.

Viewed from the anterior aspect (fig. 243) the face displays a general narrowing, the malar process showing a downward curve at its junction with the malar bone. The alveolar process around the anterior teeth reveals definite signs of rarefying osteitis and there is a spur on the septum nasi. These circumstances suggest that the individual was an habitual 'mouth-breather,' and that the contraction of the arch is the direct result of the muscular tension which is present in this condition.

(B) TREATMENT.

(1) General Considerations.—In considering remedial measures for the treatment of crowding of the teeth, there are important points which should guide us in our choice of method.

Firstly, the prevalence of *caries of the teeth* should be borne in mind, and any method which would assist the lodgment of food and increase the difficulty of keeping the mouth clean should, if possible, be avoided.

Secondly, the increasing liability to lose the teeth later on in life from parodontal disease is a reason for avoiding mechanical methods which are likely to damage the gingival margin.

The main point constantly to be borne in mind is that the **crowding of the teeth is the result of the provision of insufficient bone to allow of their normal arrangement and occlusion,** or, in other words, where the bone is defective in growth the 'arch' is too small to contain the normal number of teeth. From these premises it follows that, if a regular arch is to be secured, either the jaws must be stimulated to grow or the teeth must be reduced in number.

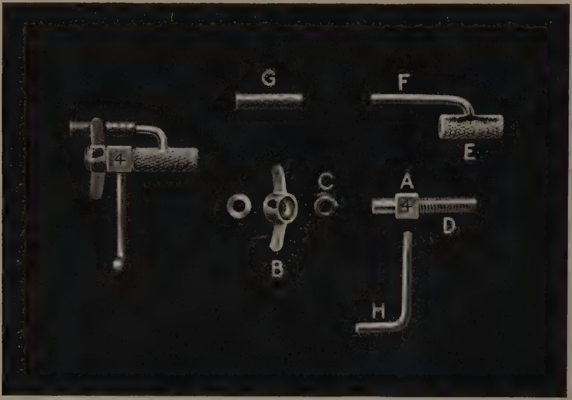
The teeth can be reduced in number by extraction ; but can the jaws be stimulated to grow ? Let us consider this question. It has already been shown that the jaws grow in a backward direction, that the only change in the bone after it is formed is a slight addition from the periosteum, and that growth is interfered with by lack of proper stimulation. If stimulation is restored, growth continues normally, but this growth does not occur in the region where the bone is deficient, namely, that part anterior to the permanent first molar. Indeed, it is difficult to see how any increase in this part can occur without interstitial growth, and there is no evidence to show that such growth occurs.

Some maintain that the jaw can be stimulated to grow and they adopt the method of treatment known as 'expansion of the arch.' This operation consists in moving all the teeth, or some of them, in an outward direction. On a previous page the movement of teeth under mechanical traction was discussed, and it was shown that, with the majority of appliances, the tooth, for all practical purposes, may be considered to swing on its apex. With 'expansion of the arch' the crowns are made to occupy a greater arch and thus obtain relief, but the relief does not extend to the apical portions of the teeth.

It is claimed that if the crowns are kept in normal apposition, the expanding of the crowns will be followed by the expanding of the roots, and further that the movement of the teeth and alveoli stimulates the growth of the defective jaw, and, indeed, of all the bones of the face, and with them the nasal and accessory cavities. It is extremely difficult to understand how this can occur, and the evidence so far advanced by its advocates is far from convincing, especially as it is quite feasible that the improvement claimed to have been made was the result of natural causes.

It is urged against 'expansion of the arch' that considerable derangement of the occlusion may result, but those who advocate this method maintain that if derangement occurs it is due to faulty manipulation.

Expansion of the arch may be carried out with either removable or fixed apparatus. A most satisfactory removable appliance is that suggested by J. H. Badcock, which consists of a split vulcanite plate actuated by an expansion screw. The screw device (fig. 244) consists of a stout wire, one end of which is threaded and works in a tube, while the other is smooth and turns stiffly in a bearing. Between the threaded and smooth portions is a square boss, and fixed to the outside of the threaded tube is a parallel guide bar. The screwed tube is attached to one half of the plate and the screw in its stiff bearing is attached to the other half of the plate, the parallel guide serving to prevent rotation of one half of the plate upon the other. The boss is turned by means of a spanner a quarter turn every other day, or, roughly, one revolution per week, a rate which gives satisfactory progress without the slightest discomfort to the patient. In this way it is possible to calculate the time necessary for a given amount of expansion. The plate is best retained by Jackson cribs, for preference on deciduous molars, the plate being cut to a knife edge on the lingual side. An arrow should be engraved on the plate to indicate the direction in which the screw is to be turned. A typical case, showing the application of the Badcock screw, is shown in fig. 245.



Left : Badcock's Screw, actual size. *Right :* Component Parts.

FIG. 244.—(A) is a square boss in the middle of the screw whereby the screw is turned. One end of this screw is smooth and revolves stiffly in the winged hub (B) in which is located the spring tension device (C); the wings are provided for the purpose of keeping the hub firmly fixed in the vulcanite. The other end of the screw (D) is threaded to correspond with the thread in the tube (E) which is knurled so that the vulcanite will hold it securely; soldered to (E) is a plain wire (F), the free end of which slides in the sleeve (G), which is closed at one end to prevent the entrance of the vulcanite, and is also knurled to give it a strong anchorage. This wire and sleeve give added rigidity to the apparatus, and, moreover, as the two halves of the plate must of necessity open parallel, it is impossible for them to revolve on one another. Fixed to one side of the square boss is a wire tag (H), which is embedded in the plaster when flasking to retain the device in position during the packing process; it is screwed out after vulcanisation.

The plain wire (F) and the sleeve (G) can be shortened if necessary, as in the case of a very narrow palate.

(For the use of this block we are indebted to Messrs. C. Ash & Sons.)

In cases where the front teeth project a most useful adjunct to the expansion plate is a flattened flexible wire arch soldered to the crib on each side. The separation of the halves of the plate draws the wire tight and exerts backward pressure on the anterior teeth. The wire should be furnished with a U-shaped bend in the vertical plane in the premolar region of each side to allow of adjustment.

The shortening of any of the incisors can be effected by means of flat wires soldered at right angles to the arch and hooked over the cutting edges. It is generally desirable to employ two such hooks in order to prevent the arch from slipping up when tightened.

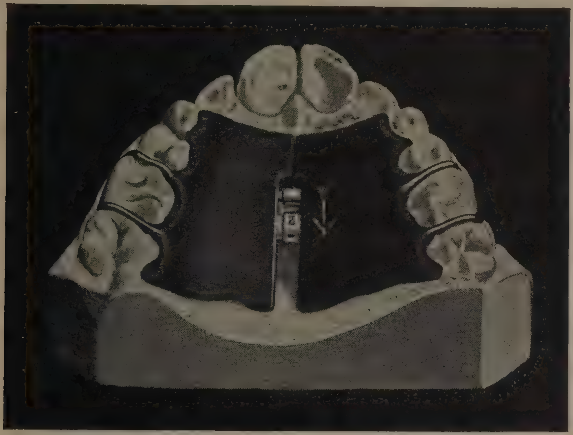


FIG. 245.

(For the use of this block we are indebted to Messrs. C. Ash & Sons.)

In Badcock's opinion the three operations of expansion, retraction, and shortening can be effected with a plate such as that described above, and, as the plate can be easily manipulated by the patient when once it has been accurately adjusted, it is specially useful for cases which are withdrawn from skilled supervision for several months at a time, *e.g.* children at boarding-school.

Of fixed apparatus for expansion of the arch E. H. Angle[1] has introduced a wire expansion arch. It consists of a wire

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[1] Though this orthodontic appliance is popularly known as the 'Angle Arch' its invention really dates back many years before the time of Angle. A labial arch used in an exactly similar manner was illustrated by Fox in England in 1806 and by Fauchard in France still earlier. The only essential difference is that instead of being fixed to anchor bands on the permanent first molars it was ligatured to those teeth.

(fig. 246), the ends being threaded and provided with friction lock nuts. The teeth used for anchorage are banded and bands are cemented to the various teeth to be moved. To the outside of each band a catch is soldered of the shape shown in fig. 247. The arch is fixed in position as follows: 'One of its screw ends is inserted in the sheath of the anchor band of one of the first molars, and the friction lock nut is seated. The arch is then sprung with the fingers into the slots in the brackets, which it accurately fits, beginning with the one nearest to the first anchor band and proceeding in regular order until the anchor band on the opposite molar is reached, when the second nut is adjusted to approximately its proper position on the arch, but neither the end of the arch nor the friction portion of the nut is at this time placed within the sheath of the anchor

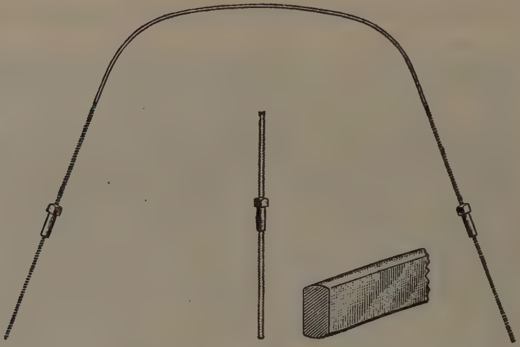

Fig. 246. (From *Dental Cosmos*, Sept. 1916.)

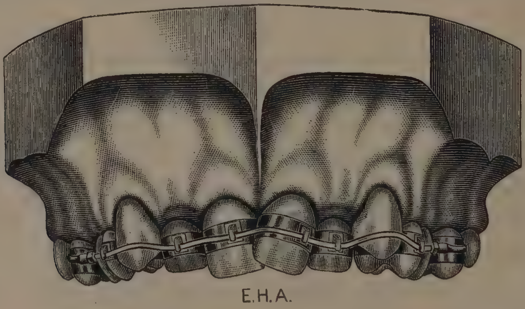

Fig. 247. (From *Dental Cosmos*, Sept. 1916.)

band. The arch is then disengaged from all the brackets, but not from the sheath of the first anchor band. Now the screw of the loose end of the arch is slipped into its sheath on the second molar band, and the nut seated. Then, beginning as before, the arch is replaced in the brackets in exactly its former relation, the last step being the proper seating and accurate adjustment of the second nut in its anchor sheath.'

'By studying the engraving (fig. 248) it will be easy to understand the manner in which the force is applied to each of the malposed teeth in order to effect not only their individual but also their collective movements. The elasticity of the arch, in this special case, operates constantly for the labial movement of the laterals, the torsional movement of the centrals, the buccal movement of the first premolars, and the lingual movement of the canines. And, as there is no waste of power through the slipping of attachments, the movement of the teeth must continue until the force from the elasticity of the arch as then bent has become exhausted, and the arch passive. Force is renewed by removing the arch and bending it to more nearly ideal form, when it is again sprung into place in the brackets and sheaths. By this means the teeth are moved continuously, until finally the ribbon arch has been given the exact form desired for the dental arch, and the teeth, through their bracket attachments, have been made to conform perfectly to it. As the

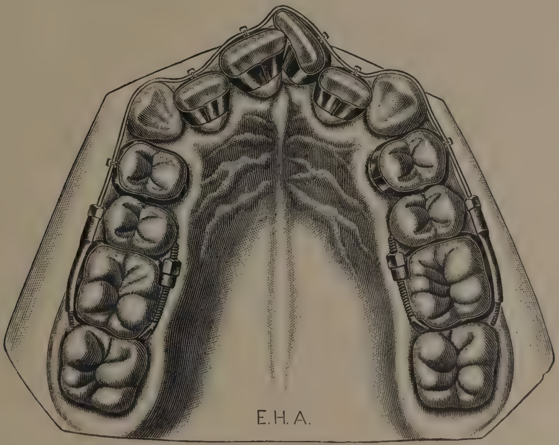

FIG. 248. (From *Dental Cosmos.*)

metal of which the arch is made is very elastic, and as the ribbon form permits much greater range of elasticity than does a round arch, the force will be continued for a correspondingly longer period before it is necessary to remove the arch and modify its form. Indeed, but two or three modifications during the whole course of treatment would probably be enough in most cases. The force is gently and almost continuously operative in exactly the right direction, without the injurious disturbance of cell function which must follow the use of any mechanism that requires frequent removal and replacement.' (Angle.)

The majority of those who favour expansion recommend that treatment should commence soon after the eruption of the incisors, the deciduous second molars being used as anchorage.

It will readily be seen that expansion entails a long period of mechanical treatment, and this is most undesirable from the point of view of the hygiene of the mouth and interference with physiological function.

Advocates of expansion claim that the facial expression is improved by the attainment of ideal occlusion, but this is after all largely a matter of personal opinion. The features in many of the illustrations of cases of expansion seem to have lost rather than to have gained in expression.

In considering the alternative treatment of crowded arches, namely, extraction, the following points can be urged in its favour :—

(*a*) Room is gained for the roots of the teeth as well as for the crowns, so that the number of teeth is commensurate with the amount of bone which growth has provided.

(*b*) Mechanical treatment is minimised and in many cases dispensed with.

(*c*) Isolation of the teeth is often obtained, and this is a point of therapeutic importance in the prevention of caries.

(*d*) The occlusion is but little deranged, and is usually considerably improved, provided that the extraction is judiciously carried out.

(*e*) The teeth invariably assume better directions.

(*f*) There is no interference with the strenuous physiological functioning of the teeth, which, in turn, promotes the efficient blood supply and development of all the other tissues associated with the act of efficient masticatory function, and should not allow of food and bacterial stasis at the gingival margin.

In the large majority of cases, therefore, extraction is far preferable to and would appear to be more rational than expansion of the arch.

When employing extraction, it is very rarely needful to remove a **first incisor** or **canine.** Occasionally, the removal of one or both the **second incisors** is necessary. Generally, a tooth posterior to the canine can be removed and a choice has to be made between the first and second premolars and the permanent first molar.

The removal of the **first premolar** has the advantage of relieving the crowding of the anterior teeth more easily than the removal of either the second premolar or first permanent molar. Its removal causes little disturbance of the occlusion of the teeth. If, however, the amount of room required is slight, there is some risk of creating too great a space between the canine and the second premolar.

Extraction of the **second premolar** ultimately provides less room than removal of the first premolar, but more room than removal of the permanent first molar. The occlusion of the teeth is only slightly disturbed, and any space that may result would be between the first premolar and permanent first molar and would be less unsightly than a space between the canine and second premolar. Removal of the second premolar has the advantage of freeing the anterior surface of the molar, and this will lessen the tendency to caries and allow any caries which may exist to be more easily treated.

Removal of the **permanent first molar** ultimately gives still less room in the front of the mouth than the removal of the first premolar, or, probably, the second premolar. The treatment necessary is also more prolonged and the occlusion of the teeth is liable to be disorganised, the mandibular second molars tilting, and the premolars, both maxillary and mandibular, rotating. As an organ of mastication the permanent first molar is most important. It possesses the largest area of crown surface and is situated in that part of the arch where the muscles of mastication work to greatest advantage. Its importance as an organ of mastication is so great, and the disturbance to the articulation caused by its removal often so marked, that it *should be removed for the relief of crowding only in cases when it is unsavable.* Under all other circumstances the choice should lie between the first and second premolars ; if both teeth are free from caries, the first should be removed if the crowding is marked; if little space is required the choice should fall on the second premolar.

A combination of extraction with expansion is occasionally useful in cases where the amount of crowding cannot be wholly overcome by expansion, and yet expansion is to some extent desirable.

The Effect of Expansion of the Arch on the Nasal Fossa.—Children suffering from nasal obstruction associated with a contracted arch are often referred by the rhinologist to the dental practitioner with the view of expanding the arch to relieve the nasal stenosis. In justification of this operation it is claimed that the median maxillary suture may be forced open by rapid expansion; the separation of the bones will then allow the septum to straighten itself and bone growth will take place at the median line, the result being that the floor of the nasal fossa is permanently widened. The claim is supported by appearances seen in radiograms before and after treatment. But such evidence is extremely unsatisfactory. The distortion of radiograms of the maxilla is well known, and there is special difficulty in taking two pictures at the same angle, which is necessary for such evidence.

In considering whether the nasal fossa may be affected by expansion of the arch, the anatomical relation of the parts must be first considered. The maxillæ have as lateral abutments the malar processes, the malar bones and the zygomatic arches with their attachments to the sides of the brain case, and these abutments are still further supported by the pterygoid processes and the vertical plates of the palate bones. They are permanent structures of comparatively hard bone, whereas the bone of the alveolar process is of far softer character. If the maxillæ are forced apart, the lateral abutments must give way, and it is not reasonable to think that they would yield before the softer bone of the alveolar process. It is, however, a clinical fact that in a few cases of marked constriction of the posterior part of the arch nasal respiration is improved by spreading the arch. M. Cryer[1] has suggested a reasonable explanation of this phenomenon. The effect of the narrow arch is to compress the tongue and force it back into the oro-pharynx. 'The tongue,' he says, 'pushes up the soft palate against the pharyngeal wall and cuts off respiration through the nose. As the tongue forces the epiglottis downwards and backwards it also interferes with oral respiration, which, however, is a little easier than nasal respiration under those circumstances; hence "mouth-breathing." ' Spreading the dental arch gives relief to the nasal obstruction by allowing more room for the tongue, which moves forward and releases the pressure on the soft palate and other structures.

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[1] *Items of Interest*, January 1913.

Oral Screens.—The use of oral screens in the treatment of these cases, with or without the extraction of teeth (dependent on the amount of room available), is often of considerable value (see p. 253).

(2) Treatment of Cases coming under Observation before the Premolars and Canines have Erupted.—When crowding is inevitable, treatment should be commenced at an early age. For simplicity of description this question will be considered under headings: (*a*) Cases where the permanent first molars are unsavable; (*b*) Cases where the permanent first molars are savable.

(*a*) *Where the Permanent First Molars are Unsavable.*

In these cases good results accrue from a line of treatment somewhat similar to the following: The permanent first molars are filled or treated in the manner best calculated to retain them until the permanent second molars have erupted. The crowding of the maxillary and mandibular incisors is then relieved by the removal of the four deciduous canines. If the teeth erupt in the normal way, the first and second premolars will come into good position, and we shall then have the following condition: the four incisors and the four premolars will be in a regular arch with a greater or less space between the second incisors and the first premolars so that the canines erupt just external to the arch; in other words, we shall have to deal with a fairly simple irregularity, namely, the canines high in the arch. To make room for the canines, the permanent first molars should be removed directly the permanent second molars are fairly through the gums. A plate to hold back the second molars should then be inserted. This plate (fig. 249) is made to cover the palate so that it comes in contact with the palatal surfaces of the incisor teeth, while to the back of it are fixed half-round gold wires which pass around the anterior and buccal surfaces of the second molars, the plate being quite free of the premolars. This form of plate retains the second molars in position and prevents them from moving forward, while at the same time it allows the premolars to fall back, partly by the action of the bite and partly by the pressure of the canine tooth, so that many cases require no further mechanical treatment—a point of no small importance.

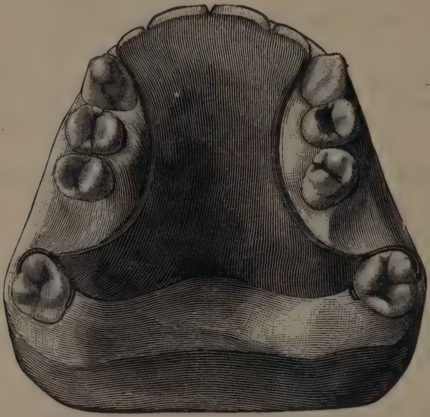


FIG. 249.

Obviously, this line of treatment will not be suitable in all cases. In some patients, the mandibular anterior teeth are so crowded at an early age that extraction of at least one of them becomes necessary. A good example of this is seen in fig. 250. Here the permanent first molars are quite unsavable, and the crowding of the mandibular incisors is excessive. This condition is due to pressure from the canines, and removal of the permanent first molars will not sufficiently relieve the pressure to allow the second incisors to assume anything approaching a normal direction. If left untreated, the mandibular canines will, in all probability, erupt in a plane anterior to the second incisors.

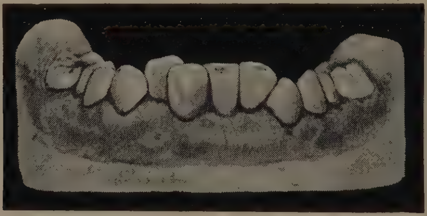


FIG. 250.

Removal of the right first incisor, however, will relieve the crowding and allow the remaining teeth to assume good directions. The result of treatment is seen in fig. 251 (see also p. 227).

Another example, which could hardly be treated on the general principles just indicated, is to be found in cases where at an early age it is clear that the maxillary permanent canine will erupt over the position of the second incisor. In these circumstances, the second incisor is forced much internal to the bite, or is so rotated that it occupies a very abnormal position. The second incisors must be extracted, and this should be done before the canines erupt. The permanent first molars should be removed before the eruption of the permanent second molars, in order that the latter may move well forward and so fill up the gap caused by the extraction of the permanent first molars. This will prevent undue spacing between the anterior teeth, which would be unsightly.

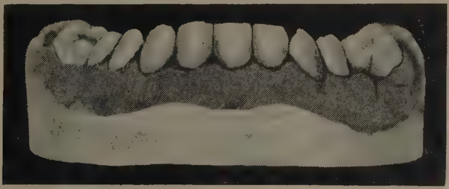


FIG. 251.

When the pulp cavities of the permanent first molars are exposed, these teeth, if retained, are an active source of sepsis. In such cases the best line of treatment is to remove the permanent first molars and the deciduous molars ; the removal of the latter teeth allows the developing premolars to travel backwards, the movement beginning before their eruption, and so relieves the crowding of the anterior teeth.

(*b*) *Where the Permanent First Molars are Savable.*

Attention should, in the first instance, be directed to the permanent first molars, and these teeth should be filled if necessary. The probable position of the permanent canine should next be determined. If the incisors are in a fairly regular line, or if the second incisor has its medial angle tilted forward, it may be assumed that the canine will erupt posterior to the incisor. If, on the other hand, the distal edge of the second incisor is projected forward, and the apex of that tooth is directed towards the median line, it may be assumed that the canine will, in all probability, erupt over the situation of the incisor, and the same may be assumed if the incisor is placed much internal to the bite, and the deciduous canine lies close to the first incisor.

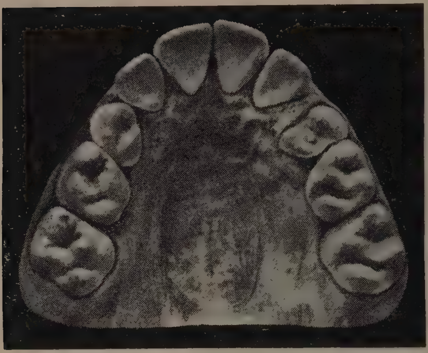


FIG. 252.—Maxillary teeth, before treatment.

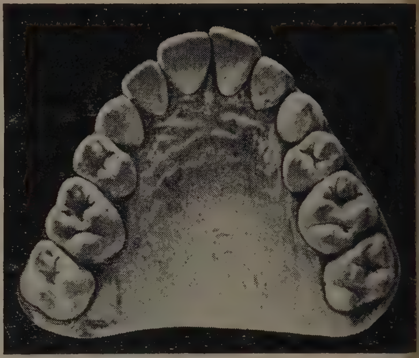


FIG. 253.—Maxillary teeth, after treatment.

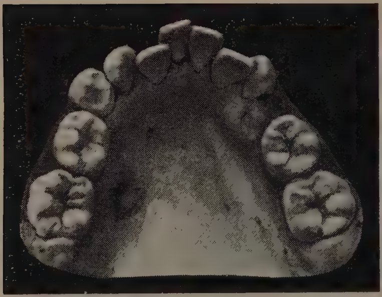


FIG. 254.—Mandibular teeth, before treatment.

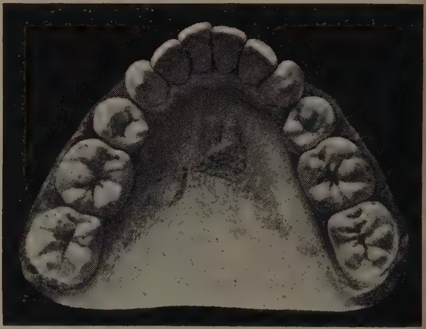


FIG. 255.—Mandibular teeth, after treatment.

Where the canine shows signs of erupting posterior to the second incisor, the treatment must depend upon the degree of crowding. If the crowding is slight, the deciduous canine should be removed and further treatment delayed until the permanent canines show signs of erupting. Either the first or the second premolar should then be removed, the choice depending upon the amount of space required for the erupting canines. In the case shown in figs. 252 to 255 the first premolars were removed immediately the canines showed signs of erupting.

If, however, the crowding of the incisors is marked, the unerupted first premolar should be removed. This operation permits the permanent canine to move backwards and so relieves the pressure on the anterior teeth (see fig. 256). It may be argued that extraction of the deciduous canine would suffice to remedy the position of the second incisor, but, if the dried specimen be carefully studied, it will be noticed that the removal of the deciduous canine will not provide room for the permanent canine.

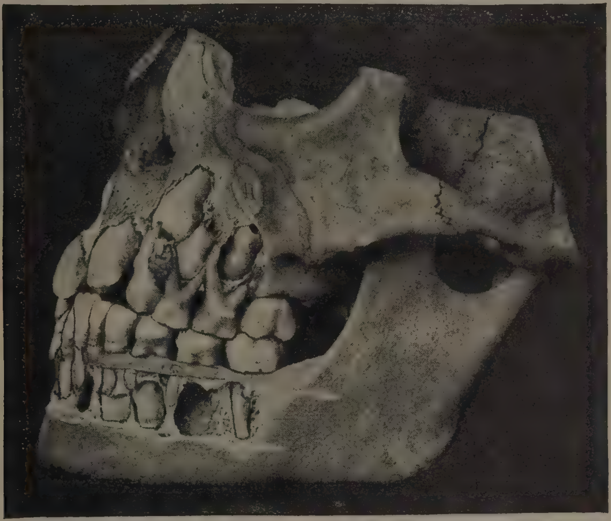


FIG. 256.—In this specimen the second incisor is placed internal to the arch with the canine partly covering the labial surface.

Treatment of crowded mouths in this manner gives excellent results. The canine moves into the space previously occupied by the premolar. Pressure on the anterior teeth is relieved, a good arch is obtained, and, what is most important, the teeth will be in excellent direction. In addition, mechanical treatment is avoided, and the occlusion of the molars and premolars remains good. Figs. 257 to 259 illustrate this method of treatment. At times, the space in the arch between the second incisor and the permanent first molar is insufficient even for two teeth, a condition shown in fig. 260. In these circumstances, the molar should be retracted until there is room for the canine and one premolar; the first premolar should then be extracted.

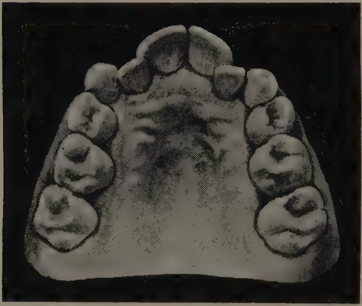


FIG. 257.—Before treatment. (In this case the second incisors had to be moved forward over the mandibular teeth.)

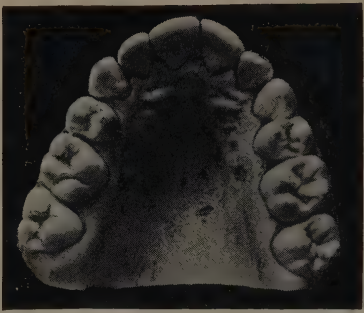


FIG. 258.—After treatment.

The advantages of the early extraction of the premolars, where serious crowding is inevitable, are :—

(1) Pressure on the front teeth is relieved and any irregularity of position which they may have assumed is easily rectified.

(2) The canines and premolars come down in a good direction and form a regular arch.

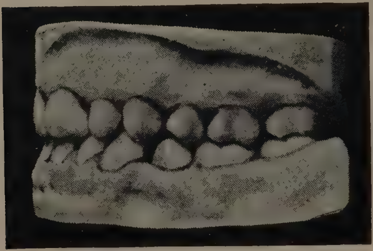
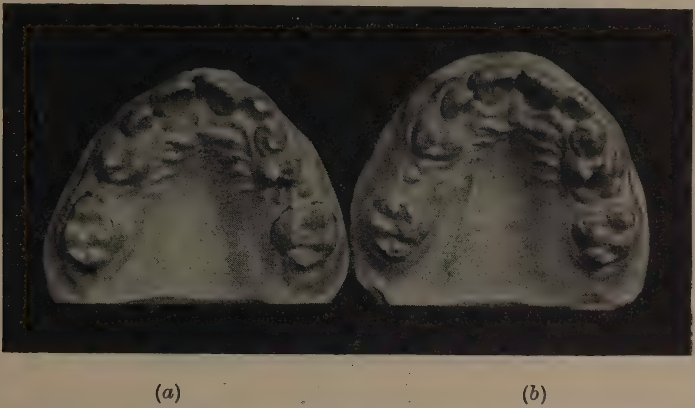


FIG. 259.—Side view, showing occlusion of premolars and molars after treatment.

Where the canines show signs of erupting over the situation of the second incisor, little advantage is gained by the removal of the premolars, and the more satisfactory treatment in such cases is to remove the second incisors before the canines erupt. A case treated in this way is shown in figs. 261 and 262.

Where only a slight degree of crowding may be anticipated, early extraction of the first premolars leads to considerable contraction of the arch in the region of the canine, and it is better in such cases to delay treatment until the canines and premolars are in position and then to remove the second premolars.

(3) Treatment of Cases coming under Observation after the Premolars and Canines have Erupted :—



(a) (b)

FIG. 260.—(a) before retraction of the first molar; (b) after retraction of the first molar.

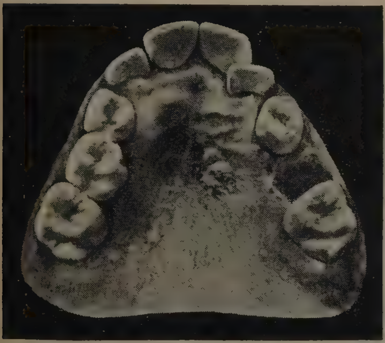


FIG. 261.—Before treatment.

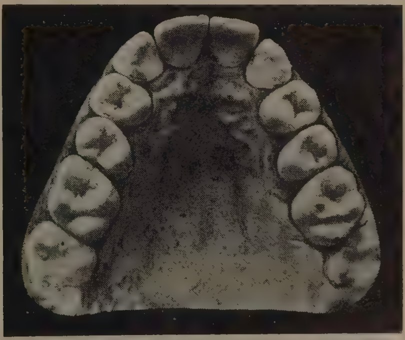


FIG. 262.—Case shown in fig. 261, after treatment.

(a) **Crowding complicated by Disease of a First Incisor.**—The treatment of such cases must depend upon—

(i) The prospect of permanently retaining the affected tooth.
(ii) The degree of crowding.
(iii) The sex of the patient.
(iv) The age of the patient.

If the pulp of the first incisor died after the root was complete there is a reasonable prospect of saving the root and crowning it, provided that no serious parodontal trouble supervenes. Under these conditions, the tooth should be saved and the crowding treated by the removal of a posterior tooth. But if the pulp died before the completion of the root the tooth should be removed.

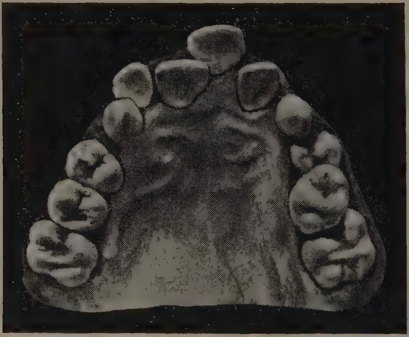


FIG. 263.

The question whether the space left by the removal of the tooth should be allowed to close up must be decided according to the merits of each case. With a boy, especially if there is considerable crowding, as shown in fig. 263, the space should be allowed to close up in order to avoid the necessity for an artificial denture—a point of importance as far as the hygiene of the mouth is concerned. With a girl, the disfigurement is of more serious import, and the space should therefore be retained by suitable means, an artificial denture being subsequently inserted.

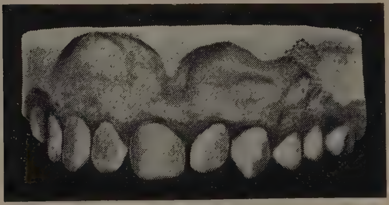


FIG. 264.—The case shown in fig. 263, after treatment.

(*b*) **Crowding which has resulted in the Protrusion of a Permanent First Incisor.**—An example of this irregularity is shown in fig. 265. The patient was aged twenty-one. The arch was expanded and the projecting incisor retracted. The result of the treatment is shown in fig. 266.

(*c*) **Crowding which has resulted in the Exclusion of Permanent**

Second Incisors from the Arch.—The advisability of sacrificing a second incisor in the treatment of crowding is constantly disputed, some practitioners even going so far as to maintain that under no circumstances can the adoption of such a course be defended. The principal argument urged against the removal of this tooth is that the canine erupts next to the first incisor and produces an unsightly appearance. It is true that a second incisor in correct position has a better appearance than a canine

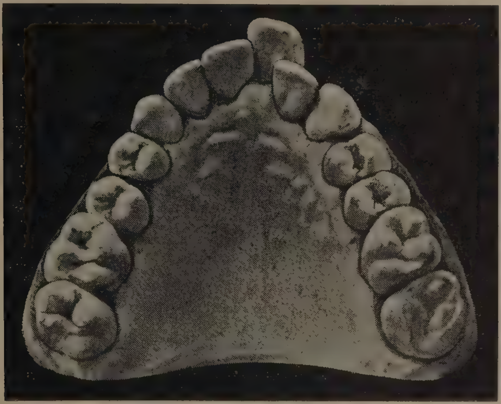


Fig. 265.

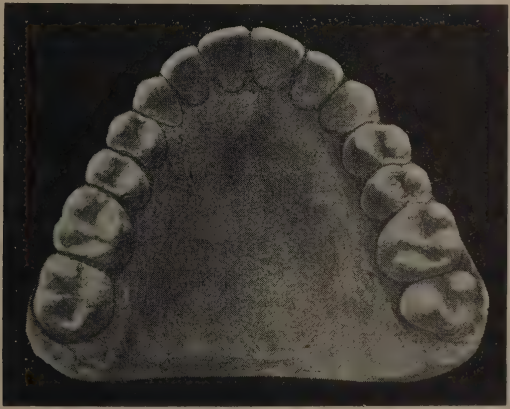


Fig. 266.

next to the first incisor, but where the extraction of a second incisor is in question, the problem to be solved may be set out as follows : Is it better to have a canine in correct alignment next to the first incisor and the premolar in apposition to the canine, or a second incisor in a maldirection, with, possibly, its cutting edge tilted forward and the canine sloping backwards, and, in all probability, short ? For example, consider the case shown in fig. 267. The second incisors are displaced inwards to a considerable extent and are in a vertical direction, the canines being but slightly external to the arch. An examination of the roots of the teeth shows the canines to be sloping towards the median line, while the roots of the first premolars take the same direction.

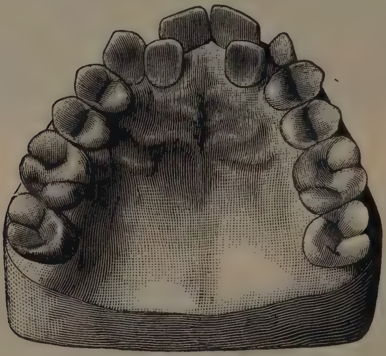


FIG. 267.

Extraction of the second incisors will effect a remedy without mechanical treatment ; the canines will come down in good direction, the first incisors will fall back and assume a more correct position, while the premolars will move forward also into a vertical position. If an attempt were made to treat the case by extraction of the first premolars, the result obtained would, in all probability, be far from satisfactory. Even with a fairly good result, and with the teeth brought approximately into a normal curve, the difference in the slope of the teeth would produce anything but a pleasing appearance. The first incisors would be forced more outward, the second incisors would have the cutting edges directed forward, that is to say, the necks of the teeth would be in a plane well posterior to the first incisors, the cutting edges of the canines would be above the level of the second incisors, and the slope backwards would be considerable.

Further, this operation would entail prolonged mechanical treatment with all its attendant disadvantages.

A row of teeth in correct alignment with the canines next to the first incisors is more sightly than the canines and the incisors in bad alignment (see diagrams in figs. 268 to 270); moreover, if the canine in its new position is adjudged to be unduly prominent, its aggressively caniniform appearance may be masked by the judicious stoning away of its tip; and it must not be forgotten that the public do not view the teeth with the critical eye of the practitioner.

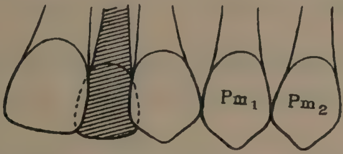


Fig. 268.—Case before treatment, showing positions of incisors and canine.

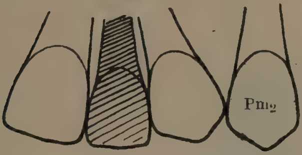


Fig. 269.—Result of treatment after removing the first premolar.

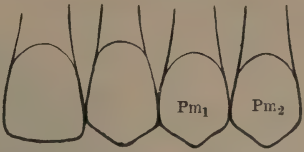


Fig. 270.—Result of treatment after removing the second incisor.

A case calling for the removal of the second incisors is shown in fig. 271.

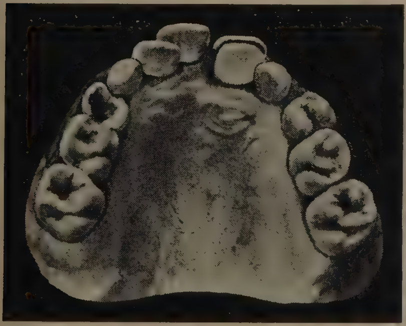


Fig. 271.

The cases which test the judgment most are those where the root of the canine is directed only slightly towards the median line and there is a possibility that the tooth may come into good direction if the premolar is removed. Under these conditions, the removal of the premolar may be the wiser course.

Sometimes the second incisors are displaced to such an extent that their extraction is imperatively called for, and the permanent first molars are also quite unsavable. Under such conditions, the molars must be removed, if possible before the permanent second molars erupt; the latter will then come forward and, in a great measure, prevent a backward movement of the premolars, which would probably leave unsightly spaces between the front teeth.

(*d*) **Crowding which results in the Displacement of the Canine either External or Internal to the Arch.**—The displacement of a canine external to the arch is the commonest result of a crowded condition of the teeth. A typical example is shown in fig. 272.

In dealing with this irregularity, the importance of the canine in the dental arch should be borne in mind. It is the longest and probably the strongest and most highly developed tooth in the arch, in which it forms a kind of keystone. Its prominence assists to give character to the face; and its extraction leaves a depression near the ala of the nose and may give an appearance of increased age by accentuating the naso-labial fold. These facts, and the flattening of the arch produced by its removal, tend to rob the face of expression. For these reasons it should be retained whenever practicable. The choice of treatment usually lies between the removal of the canine or, alternatively, the removal of the second incisor, a premolar, or the molar. The removal of the canine should be regarded as the last resource.

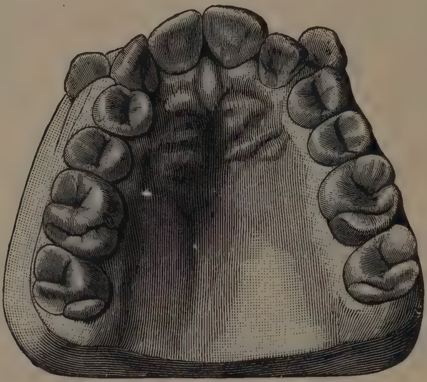


FIG. 272.

Before selecting the tooth for removal the position of the root of the canine should be ascertained. If the root has a direction forward, the removal of a posterior tooth will cause the canine, when it erupts, to slope backwards to such an extent as to present an unsightly appearance, whereas if the second incisor is extracted the canine will erupt fairly straight. If the root of the canine is directed backwards a premolar or molar should be removed. The figs. 273 to 276 illustrate these points.

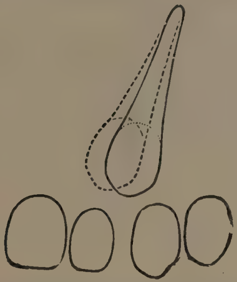


FIG. 273.—Showing the root of the canine lying over the premolar. The dotted lines represent the direction the canine will take if the second incisor is extracted.

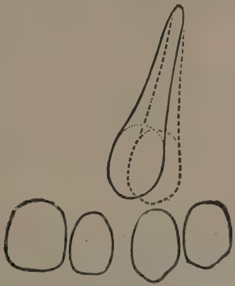


FIG. 274.—Showing the root of the canine lying over the premolar. The dotted lines represent the direction the canine will take if the premolar is extracted.

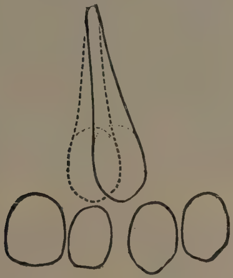


FIG. 275.—Showing the root of the canine lying over the second incisor. The dotted lines represent the direction the canine will take if the incisor is extracted.

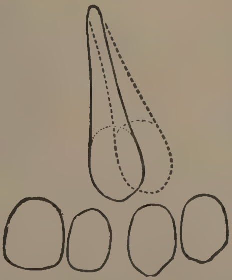


FIG. 276.—Showing the root of the canine lying over the second incisor. The dotted lines represent the direction the canine will take if the premolar is extracted.

Irregularities of the canines seldom require mechanical treatment, especially if extractions have been made with due regard to the occlusion. Nature will frequently overcome the difficulties unaided, and it is usually advisable to postpone further treatment for a period of from three to six months after extraction ; but if the tooth does not then appear to be moving into position, it should be mechanically assisted, and one of the simplest methods in vogue is that shown in fig. 181.

In removing a premolar for the relief of a crowded canine attention must be given to the position of the corresponding mandibular premolar, for the latter may be placed in such a position as to prevent the maxillary canine from coming correctly into line. In certain rare cases, however, extraction of the canines is quite justifiable, especially where, with the arch regular and the occlusion good, the second incisors are close to the premolars and the canines erupt outwards almost at right angles, as seen in fig. 277.

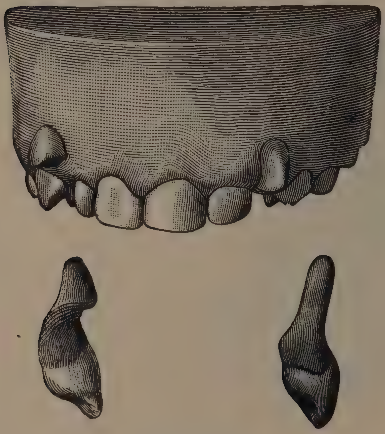


Fig. 277.

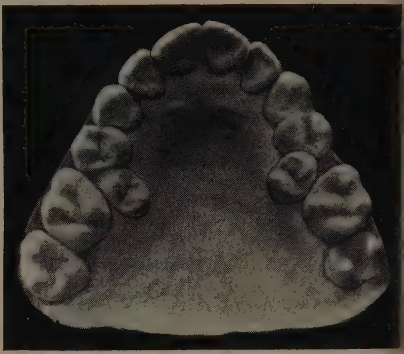


Fig. 278.

(*e*) **Crowding which results in Displacement of Premolars.**—Displacement of the second premolar internal to the bite frequently results from crowding. When this occurs, the second premolar has usually erupted after the canine. Fig. 278 is an excellent example of this type of irregularity. The treatment would be the removal of the abnormally placed premolars, but if the molars were unsavable, it would be necessary to remove them and bring the premolars into the line of the arch.

(*f*) **Crowding which results in the Displacement of a Mandibular Incisor.**—An irregular arrangement of the mandibular incisors caused by crowding is not uncommon, the canines being mainly responsible for the irregularity. The canines are normally developed external to the incisors, the crowns often slightly overlapping the labial surfaces of the second incisors, and where there is insufficient room for their development, the canines encroach on the incisor region and so cause irregularity.

Crowding of the anterior mandibular teeth is not so important from an æsthetic point of view as crowding of the anterior maxillary teeth. The most effective treatment when the crowding is severe is to remove an incisor. In selecting the incisor to be removed, the following points should be considered :—

(i) A first incisor should, if possible, be extracted in preference to a second incisor for reasons of symmetry ; besides, it is usually the narrower tooth, and its loss leaves less space to be taken up by the ultimate movements of the adjacent teeth. If a second incisor be removed, the canine on one side will be adjacent to a first, and on the other side to a second incisor.

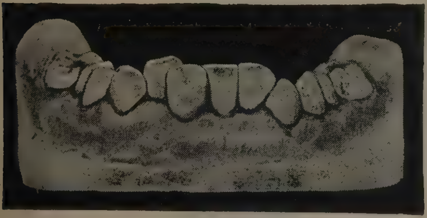


Fig. 279.

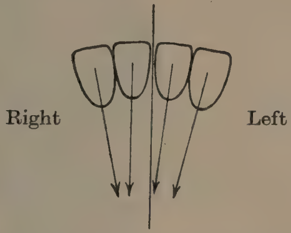


Fig. 280.—Diagram showing directions of the roots of the incisors.

(ii) An outstanding tooth should be removed in preference to one instanding, as the latter will be more easily brought into correct line, the pressure of the tongue being more powerful than that of the lower lip.

(iii) The direction of the roots of the different teeth is important. The tooth selected should permit of the remaining incisors assuming a vertical direction. For instance, in the case shown in figs. 279 and 280, removal of the right first incisor would allow the remaining teeth to assume vertical directions, but removal of the left first incisor would result in the right first incisor assuming a distinct slope.

(*g*) **Mandibular Canines.**—Displacement external and internal to the arch may occur, the former being more common. The principles of treatment are similar to those for maxillary canines.

The removal of a mandibular canine does not mar the expression of the features to the same degree as the removal of the maxillary canine. The mandibular canine should be removed in cases where it is much external to the arch with the first premolar and the second incisor in good alignment.

(*h*) **Mandibular premolars** placed external or internal to the arch (see fig. 281) require removal.

(*i*) **Cases involving the Malposition of Several Teeth.**—The treatment of general irregularity of the teeth will depend, to a great extent, upon the general condition of the teeth. If the permanent first molars are unsavable their removal is indicated, but opinions differ as to the proper time to remove them in order to obtain the best results. From examination of mouths in which there is an absence of crowding of the teeth but the permanent first molars have been removed, it would appear that the best results are obtained when these teeth are removed before the eruption of the permanent second molars, and after the eruption of the premolars, as good spacing between the anterior teeth is then obtained and there is far less tilting of the mandibular permanent second molars (fig. 282). A strong objection to removing the permanent first molars at this period, in cases of crowding, is that the permanent second molars, especially the maxillary, tilt and move forward and encroach on the space required for the backward movement of the anterior teeth. In cases where the teeth are crowded the extractions should be delayed until the appearance of the permanent second molars. Many practitioners advise postponement of extraction until the permanent second molars are fully erupted and in good occlusion in order to minimise the tilting of the mandibular permanent second molars, but if the operation is thus delayed the crowding will be less easily remedied, as the teeth will have become more firmly implanted

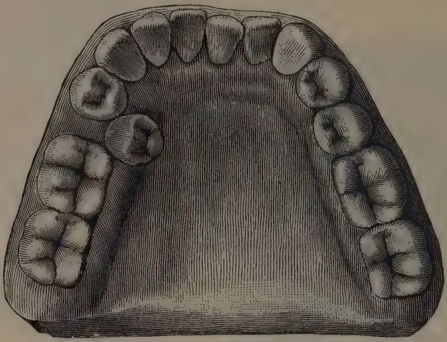


FIG. 281.

and the irregularity of the anterior teeth more pronounced. On the whole, therefore, it seems advisable to remove the permanent first molars as soon as the permanent second molars are sufficiently through to allow of the application of mechanical means for holding them back (see fig. 249). In this way the crowding can be relieved at an earlier period, and the tilting of the second permanent molars is, to a great extent, prevented.

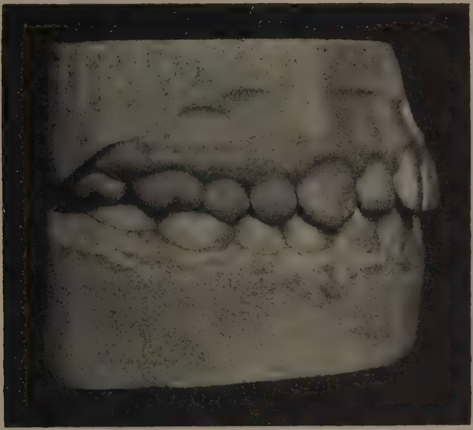


Fig. 282.—Models of a case in which the permanent first molars were removed for caries before the eruption of the second molars.

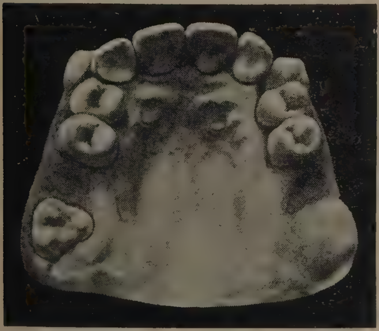


Fig. 283.—Before treatment.

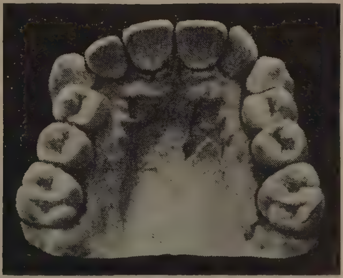


Fig. 284.—After treatment.

The ill-effects of removing the permanent first molars, in cases of crowding, before the permanent second molars erupt, are seen in figs. 283 and 284.

A certain number of cases come under treatment in which the mandibular permanent first molars have already been removed and no attention has been paid to the maxillary teeth. The following case is instructive [1] :—

Owing to the removal of the mandibular permanent first molars a year previously, the permanent second molars have travelled forward and partly articulate with the maxillary permanent first molars ; the maxillary left second premolar is displaced inwards, but otherwise there is practically no crowding (see figs. 285 to 291). Some of the maxillary incisors have been attacked by caries on the approximal surfaces. The maxillary left first molar has a cavity on the masticating surface, while in the maxillary right first molar a cavity has been successfully treated on the anterior surface.

The occlusion of the teeth on the right and left sides is diagrammatically shown in figs. 286 and 287.

On the left side it will be noticed that the maxillary second premolar is internal to the arch. The occlusion between the maxillary first premolar and the mandibular premolars is not good. The maxillary first molar is prevented from moving forward by the second premolar and is only half opposed, while its posterior cusps prevent the mandibular second molar from moving forward. The maxillary second molar is erupting, hence a posterior force is present. Extraction of the second premolar will allow the maxillary first molar and the mandibular second molar to move forward, and so lead to an improvement in the occluding surfaces. On the right side it will be seen that the anterior plane of the maxillary first premolar only partly occludes with the posterior plane of the corresponding mandibular tooth, and that there is no occlusion with the anterior plane of the mandibular second premolar as there ought to be. Only half the anterior plane of the maxillary second premolar is used. The maxillary first molar is only partly in occlusion with the mandibular second molar, and this prevents the latter moving forward. The maxillary second molar is erupting. Extraction of the maxillary second premolar would permit the maxillary first premolar to move backwards and occlude correctly with the mandibular premolars. The maxillary first molar and mandibular second molar would move forward and the occlusion be improved. The maxillary second premolars were removed, and the result is seen in figs. 289 and 291.

Where there is marked crowding in the maxilla, with the maxillary permanent first molars savable and the mandibular permanent first molars unsavable, the best results are to be obtained by removing the mandibular permanent first molars before the eruption of the permanent second molars. The latter will then come forward and occlude well with the maxillary teeth. The

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[1] The following cases are fully described with the view of showing the various points to be considered when adopting extraction for the relief of crowded conditions of the mouth.

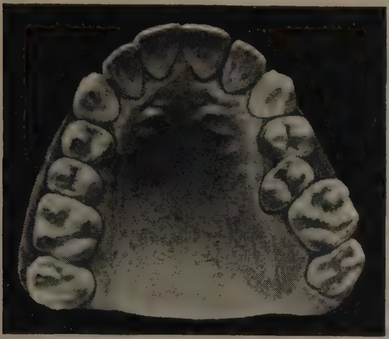

FIG. 285.

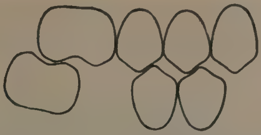

FIG. 286.—Right side.

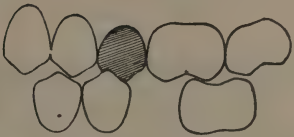

FIG. 287.—Left side.

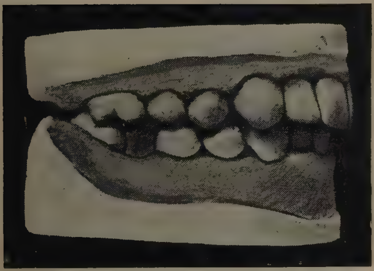

FIG. 288.
Before treatment. Right side.

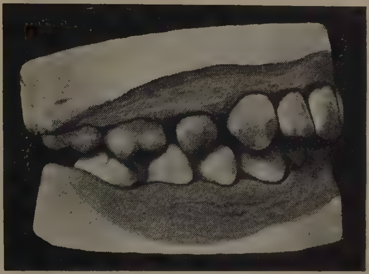

FIG. 289.
After treatment. Right side.

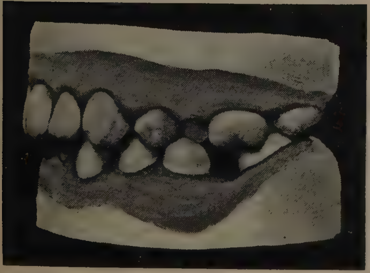

FIG. 290.
Before treatment. Left side.

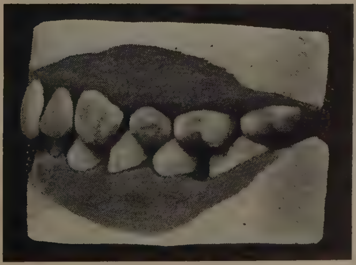

FIG. 291.
After treatment. Left side.

maxillary first premolars should be removed, if possible, before the canines erupt. In this way, the crowding of the maxillary teeth can be correctly overcome and a good articulation obtained. A case thus treated is shown in figs. 292 to 295.

Second premolars should, as a rule, be removed where the crowding is not great. Should a space remain, it will not be so noticeable as if the first premolar had been extracted. Removal of the second premolars is indicated where caries is present on the anterior approximal surfaces of the permanent first molars.

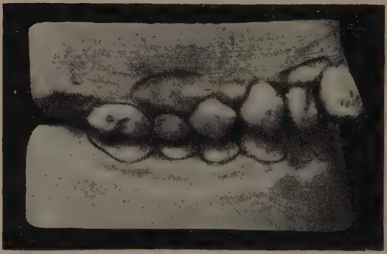

FIG. 292.
Before treatment. Right side.

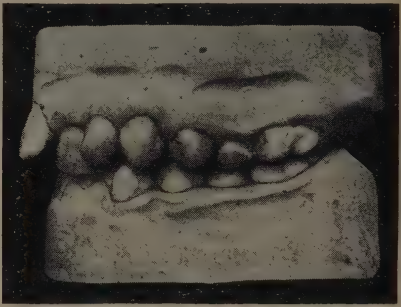

FIG. 293.
Before treatment. Left side.

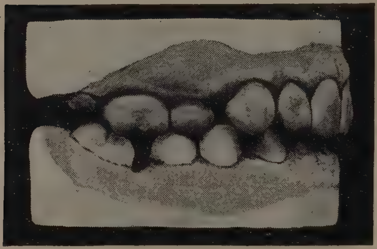

FIG. 294.
After treatment. Right side.

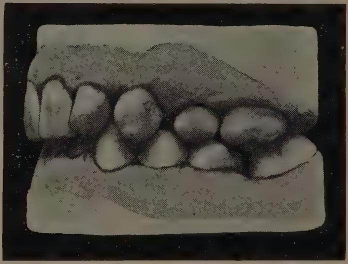

FIG. 295.
After treatment. Left side.

The following is an instructive case requiring the removal of the maxillary second premolars :—

The patient was a girl aged fifteen. The maxillary front teeth were crowded and irregular (fig. 296). The mandibular teeth presented a good arch. An examination of the premolar and molar occlusion on the right side showed that only portions of the posterior planes of the first and second premolars were in contact with the mandibular teeth (fig. 298). This is diagrammatically shown in fig. 302. Removal of the maxillary first premolar would allow the second premolar to come forward and occlude with both mandibular premolars and at the same time allow room for the canine to move backwards, but the second premolar on this side was carious. With removal of the second premolar the

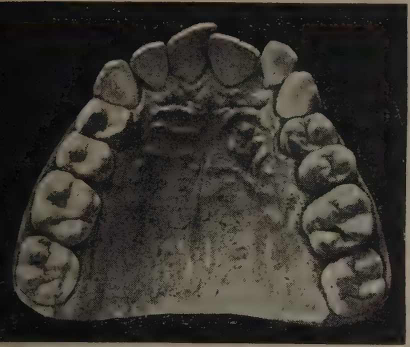

FIG. 296.—Before treatment.

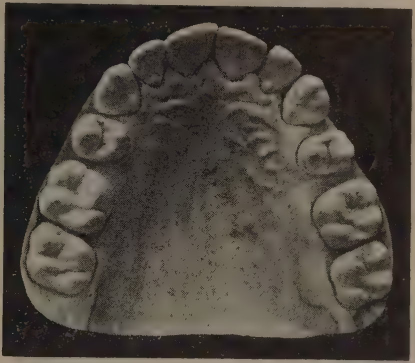

FIG. 297.—After treatment.

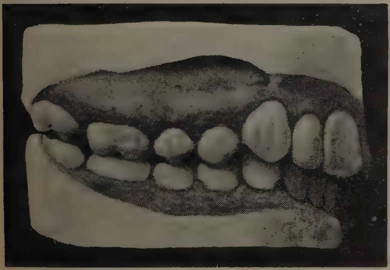

FIG. 298.—Right side. Before treatment.

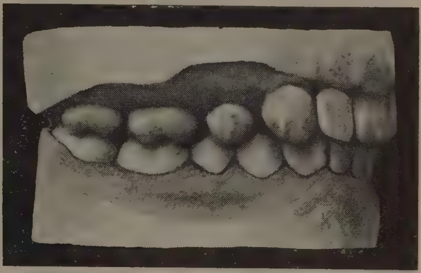

FIG. 299.—Right side. After treatment.

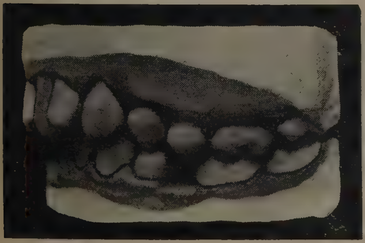

FIG. 300.—Left side. Before treatment.

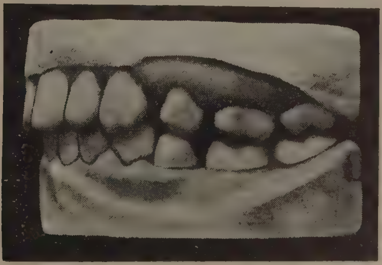

FIG. 301.—Left side. After treatment.

first premolar could be brought back and made to articulate satisfactorily with the mandibular premolars, and the crowding of the front teeth would be overcome. On the left side (fig. 300) the mandibular second premolar had not erupted, and there were no signs of its presence. The maxillary second premolar, therefore, formed but a small portion of the masticating area and was consequently the tooth to remove.

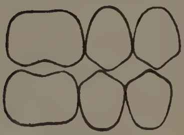

FIG. 302.

The treatment consisted in the removal of the maxillary second premolars. The first premolars were then brought back by mechanical means. A vulcanite plate capping the molars and premolars was made, half-round gold wires being attached to the sides and arranged so as to bring pressure on the medial angles of the right first and the left second incisors. Opposite the palatal aspect of the left first incisor, a wedge of compressed wood was inserted and the tooth brought forward. By a similar arrangement, the distal angle of the right tooth was driven forward. A retention plate was used for twelve months. The result is seen in figs. 297, 299, 301. A regular arch has been obtained. The occlusion of the teeth on the right side has been improved. On the left side the occlusion has not been impaired, and will be improved when the molars move forward.

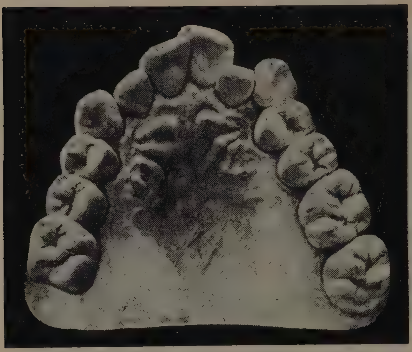

FIG. 303.—Before treatment.

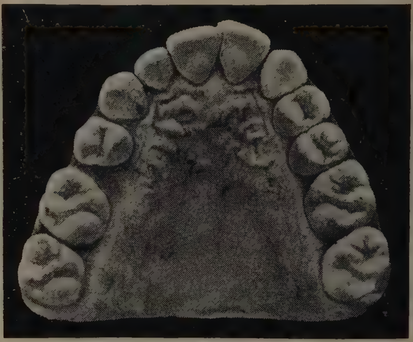

FIG. 304.—After treatment.

In a few cases the removal of a second incisor on one side and a premolar on the other may be necessary. The following is an example :—

In this patient, a girl, the anterior maxillary teeth were crowded, the second incisor on the left side being internal to the arch (fig. 303). In the mandible, the arch was regular, but the first incisors were absent. The teeth were free from caries. An examination of the roots of the left first and second incisors and canine showed that the root of the first incisor sloped in a backward direction and was placed in a plane anterior to the root of the second incisor, and also overlapped that of the right first incisor. The canine root was very slightly backward and was also in a plane anterior to the second incisor. The positions of the three teeth are shown in fig. 305. The occlusion of the premolars and molars was good (fig. 306). On the right side, the root of the canine was directed backwards, and the crowding of the incisors was slight. The relation of the maxillary to the mandibular premolars and molars is shown in fig. 308. In considering treatment, the main difficulty was the marked crowding on one side and the slight crowding on the other. On the left side, two courses were open : (*a*) the removal of the first premolar, or a posterior tooth ; (*b*) the removal of the second incisor. If removal of the first premolar were carried out, the canine would need to be retracted and the second incisor pushed out. This operation would probably result in the canine assuming a very sloping direction and being short, the second incisor having its cutting edge tilted forward, while the first incisor would be driven still more across the median line. In addition, the treatment would require the prolonged use of a plate, and there would be a

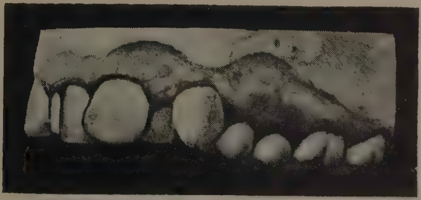

FIG. 305.

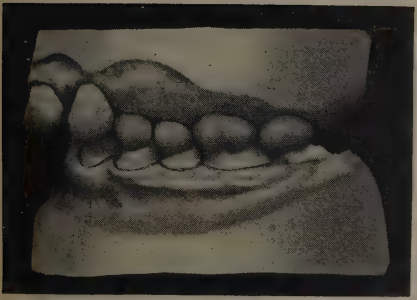

FIG. 306.—Left side. Before treatment.

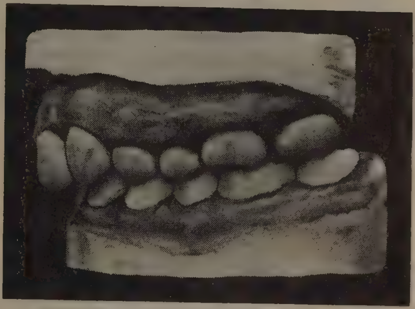

FIG. 307.—Left side. After treatment.

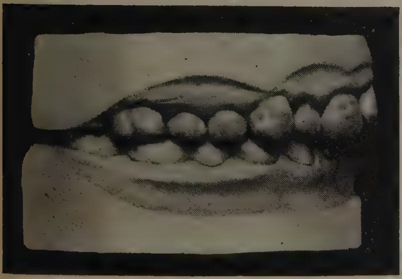

FIG. 308.—Right side. Before treatment.

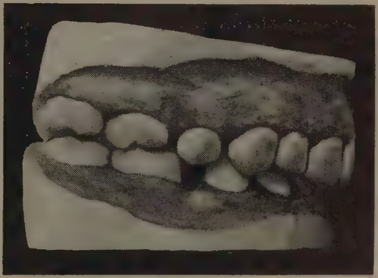

FIG. 309.—Right side. After treatment.

constant tendency to relapse. On the other hand, removal of the second incisor would permit the first incisor to fall back and assume a more vertical direction, and the canine would move forward and fill up the gap. There would be true relief of the crowding (both roots and crowns), and no tendency to relapse. On the right side but little room is required, and removal of the second incisor to correspond with removal of the left tooth is out of the question. The treatment, therefore, resolves itself into the removal of a premolar or a molar. The molar is free from caries, so a choice must be made between the first and second premolars. Removal of the first premolar would, in all probability, result in a gap between the canine and the second premolar. Removal of the maxillary second premolar alone and retraction of the first premolar would also leave a gap, because the first premolar would eventually occupy the same position as the second premolar. Removal of the mandibular second premolar as well as the maxillary second premolar would allow the first premolars, maxillary and mandibular, to move back sufficiently to overcome the crowding of the incisors and canine, while the molars would move forward and assist in filling up the gaps. In addition, mechanical methods would be avoided. Treatment consisted in the removal

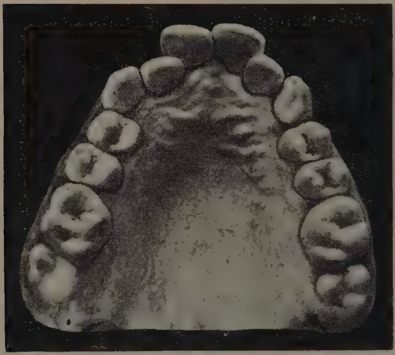

Fig. 310.—Before treatment.

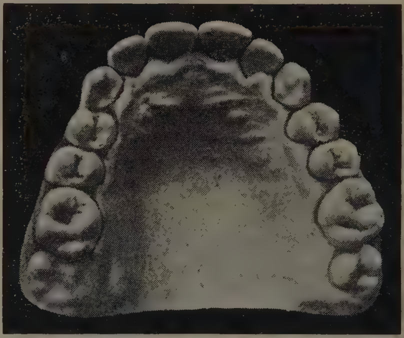

Fig. 311.—After treatment.

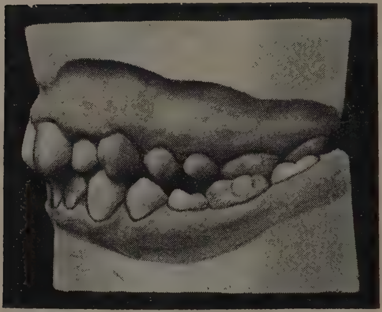

Fig. 312.—Before treatment.

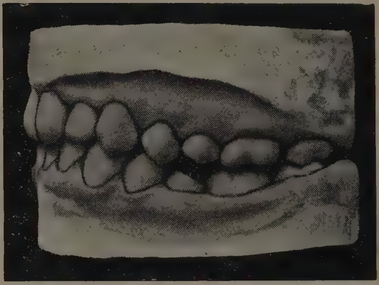

Fig. 313.—After treatment.

of the maxillary left second incisor and the maxillary and mandibular right second premolars. The result of treatment is seen in figs. 304, 307, and 309. On the right side there is a slight gap between the premolar and canine, but when the case was last seen the molars had moved still more forward and had considerably improved the occlusion of the maxillary premolar with the mandibular premolar and permanent first molar. With the advent of the third molars, the space will, no doubt, be entirely closed.

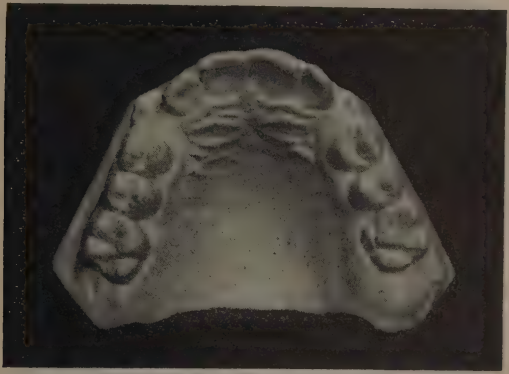

FIG. 314.—Before treatment.

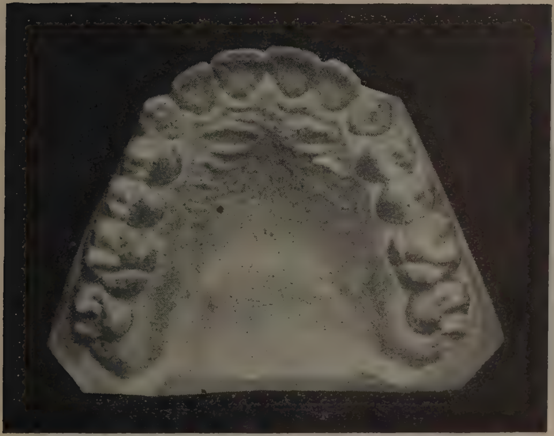

FIG. 315.—After treatment. The permanent second molars have erupted.

The operation of expansion may be adopted in cases where the general lie of the premolars and molars is inwards, and where the articulation and the personal appearance of the patient will be improved by the operation. A case treated by expansion is shown in figs. 310 to 313.

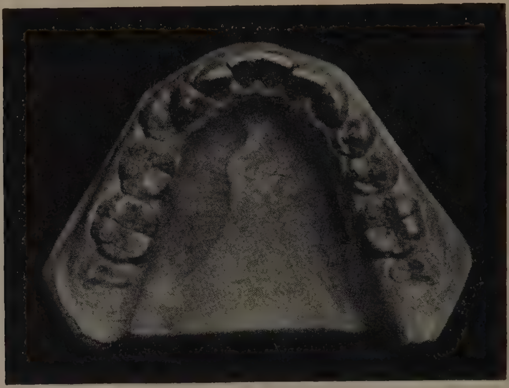

FIG. 316.—Before treatment. The second molars have partly erupted.

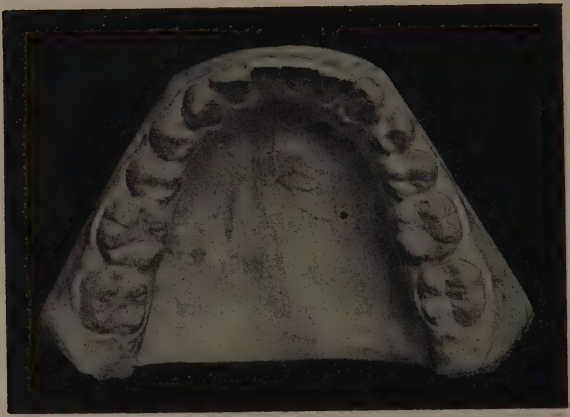

FIG. 317.—After treatment. The second molars have completely erupted.

Figs. 314 to 321 are the models of a case of crowding of the teeth under the care of G. Northcroft, which he treated by means of expansion. The child was breast-fed for three months and then hand-fed, a boat-shaped bottle being used. No adenoids. The first models of this patient were taken at the age of 8 years 4 months, and showed slight crowding of the maxillary and mandibular anterior teeth. As the condition did not improve and the occlusion was getting worse, it was decided at the age of 10 years 9 months to adopt operative treatment. From the models shown in figs. 318 and 320, it will be noticed that the molar occlusion is correct on the left-hand side, but on the right-hand side the maxillary

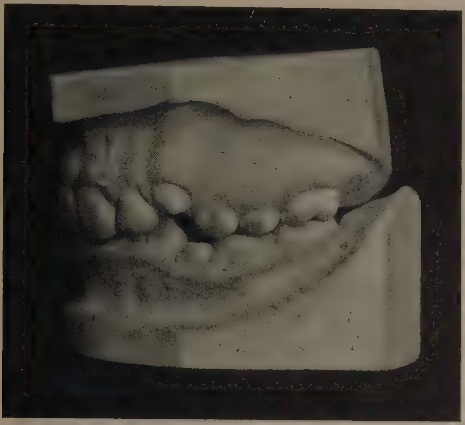

FIG. 318.—Left side before treatment.

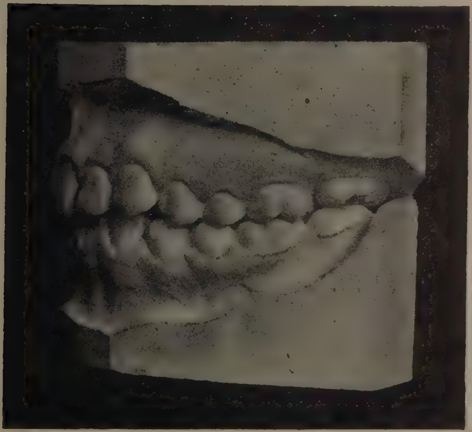

FIG. 319.—Left side after treatment.

permanent first molar is in advance of its normal position and presents a flush occlusion. The treatment consisted in applying expansion arches to the maxillary and mandibular teeth, combined with inter-maxillary reciprocal traction. Pressure was brought to bear on the maxillary permanent first molars, and a band and spur applied to rotate the maxillary left second incisor. At the end of a little over four months the appliances were removed and vulcanite retention plates inserted. The models, shown in figs. 315, 317, 319, and 321, represent the case a year and three months after completion of treatment.

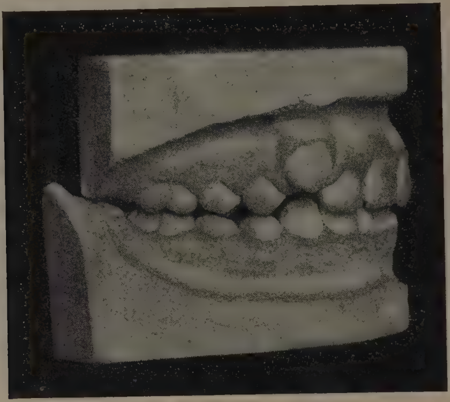

FIG. 320.—Right side before treatment.

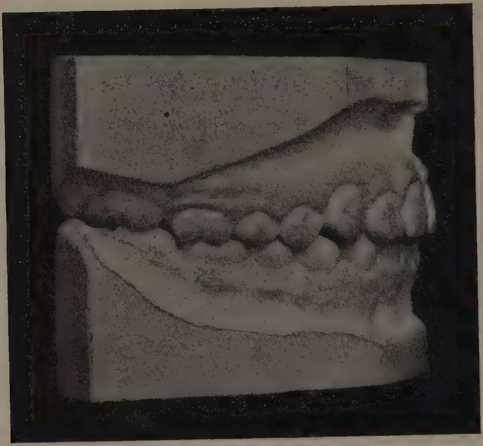

FIG. 321.—Right side after treatment.

The following are the comparative measurements of the case when seen at 10 years 9 months and when completed :—

	10 yrs. 9 mths.	Completion of treatment.
Width between $3\vert 3$ at tips	35·5 mm.	37 mm.
,, ,, $4\vert 4$ at gum-line	24·6 ,,	27 ,,
,, ,, $5\vert 5$,, ,, ...	30·5 ,,	33 ,,
,, ,, $6\vert 6$,, ,, ...	36 ,,	38 ,,
Depth of palate at $6\vert 6$ from gum-line	11 ,,	15 ,,
Length of jaw from palatal sulcus of molar to $1\vert 1$	35 ,,	36 ,,
The combined distances between $4\vert 4$ and $2\vert 2$ are	6·5 ,,	12·9 ,,

The average expansion being about 2 mm.

General Conclusions and Axioms.

Certain general conclusions with regard to the treatment of any given orthodontic case may now be made.

The objects of orthodontic treatment are twofold and are in the order of their importance. (i) The provision of a healthy mouth. (ii) The improving of the appearance. The former is done by making the teeth entirely functional, and as self-cleansing and efficient as possible ; these in turn are brought about by relief from crowding and efficient occlusion.

The age at which treatment is sought.—There is a growing tendency for cases to come for treatment at an earlier age than in the past. The advantages of early treatment are inestimable. The control of tooth movements, recognition and removal of adverse factors and habits, and provision of ample time for such operations as expansion are very important ; the provision of room for the permanent teeth before they erupt makes their ultimate correct alignment and occlusion far more of a physiological process than it otherwise would be.

Normal occlusion, though desirable, is by no means a necessity for efficient mastication ; cheek teeth often function equally well when the molar occlusion is pre- or post-normal.

Making room for the relief of crowding.—Room may be made in two ways : by expansion and by extraction.

Expansion should only be used when comparatively little extra room is required, and its most satisfactory results are seen in cases where it has been done at a relatively early age, and slowly. In the past expansion has often been overdone and/or has been done at too late an age ; when this has occurred the amount of bone provided is insufficient to support the masticatory apparatus present, the function of which is thereby impaired and so gives rise later on to parodontal disease. It entails the disadvantages of the use of mechanical apparatus which are chiefly concerned with interference with strenuous physiological functioning and its beneficent effects, and food stasis, but are also concerned with cost and time occupied by treatment.

Extraction is best when much room is needed. By leaving functioning teeth only, and doing away with stagnation areas, it immediately increases and improves physiological function; in cases brought for treatment at later ages it is frequently the only rational treatment by which room may be provided; the masticatory apparatus left is always such as the bone provided may support; frequently, particularly when the extractions are done early, no other treatment is necessary. Cost and time occupied by treatment are reduced to a minimum.

Extractions, particularly in the premolar region, should often be symmetrical in either jaw and frequently in both[1]; if this is not done, in the former case the result may be an obvious and unpleasing asymmetry, and in the latter the locking of occlusal planes may prevent the desired movements of teeth unless mechanical means be used. As a general rule the tooth nearest the one the irregularity of which it is desired to correct should be the one to be lost, but there are exceptions.

If room be conveniently provided for a crowded tooth to erupt into, it almost invariably comes into the desired position with no mechanical treatment whatever.

In a crowded mouth, the factors which largely govern whether the effects of the crowding will ultimately be most obvious in the incisor, canine, or premolar region are the order in which the canines and premolars erupt.

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[1] This does not necessarily mean that the corresponding teeth on each side should be removed; it may mean the removal of one cusp unit on each side.

CHAPTER VIII

Abnormalities of lack of occlusion of many teeth.

Superior Protrusion—Inferior Protrusion—Open Bite—Abnormalities due to Congenital Defects of the Jaw.

(A) SUPERIOR PROTRUSION.

THE term 'superior protrusion' is applied to that type of irregularity in which the maxillary front teeth project abnormally. In most cases of superior protrusion the maxillary teeth project beyond the lower lip which passes behind them and, the upper lip failing to cover them, they remain partly or wholly exposed.

Superior protrusion is, however, a relative term, because it can readily be seen that a lack of development of the mandible may also produce a condition where the maxillary teeth are too far in advance of the mandibular teeth, but that the fault may lie with the mandible rather than the maxilla.

Dependent on where the fault is considered to lie, some of these cases are called superior protrusion and others inferior retrusion. It is not always easy to decide where the fault does lie, and the deciding factor in considering this, and the appropriate nomenclature in each case, must rest on the appearance of the degree of development of the maxilla and mandible when the child is seen in profile.

Protrusion of the maxillary incisors may occur either in the permanent or the deciduous dentition. In the deciduous dentition the maxillary incisors are normally placed almost in a vertical position and in close contact with the labial aspects of the mandibular incisors. A slight forward movement of the maxillary incisors sometimes occurs and may escape notice, but it is important because it foreshadows protrusion in the permanent series. In their crypts the maxillary permanent incisors have their cutting edges directed a little outwards, and a forward movement of the deciduous arch tends to increase the outward direction of the permanent incisors, with the result that in the process of erupting the teeth are liable to pass in front of the lower lip. Should this occur, the irregularity is accentuated by the twofold action of the lower lip, which tends to force the maxillary teeth forward and the mandibular teeth backward.

Etiology and Pathology.

Cases of superior protrusion may be divided into groups, each of which possesses clearly defined characteristics.

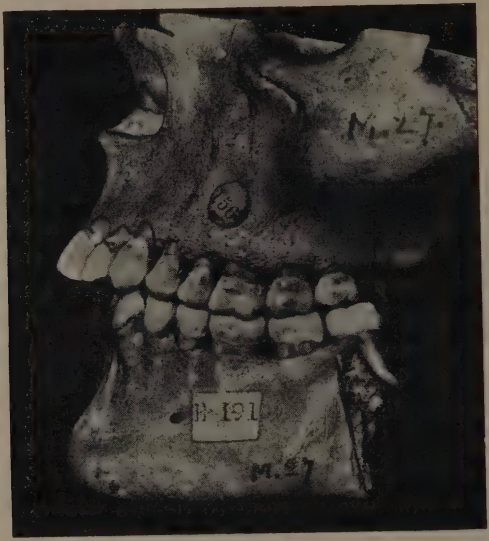

Fig. 322.

Group 1.—The jaws are well developed and there are no indications of deficient growth, though the maxillary arch may be rather narrowed in the premolar region ; the maxillary incisors are spaced ; the mandibular incisors may present a normal curve, in which case the cutting edges are free from the mucoperiosteum of the palate ; or the arch may be flattened, with the cutting edges touching the palate. The occlusion of the mandibular molars may be normal or slightly postnormal. A view of the profile (fig. 322) shows that the chin is well formed. The postnormal occlusion of some cases might be taken to imply that either the maxilla is over-developed or the mandible under-developed, but in this group of cases it is probable that neither of these is primarily at fault. The cause probably lies in the persistent and prolonged habit of thumb-sucking or sucking the lower lip or such object as a dummy-teat comforter, and the projection of the maxillary incisors is usually traceable to such cause. It may readily be conceived that forcing the mandible back in this manner may cause postnormal occlusion in severe cases; the lateral pressure of the canines in addition to backward pressure on the mandibular incisors causes their displacement upwards, so that they assume a fan-shaped arrangement.

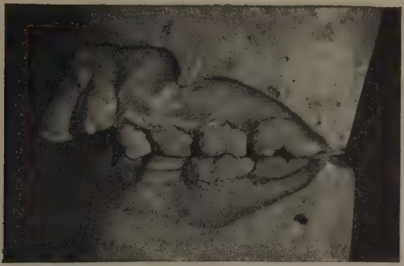

Fig. 323.—A case of superior protrusion due to sucking the lower lip.

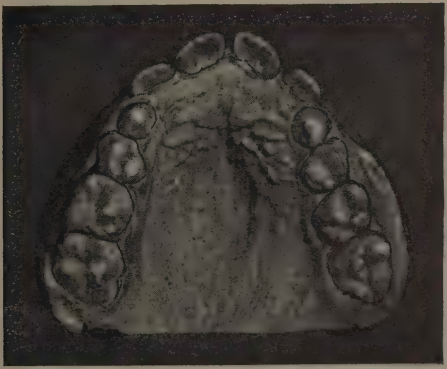

Fig. 324.—View of the maxillary teeth of the case shown in fig. 323.

In figs. 323 to 325 are shown the models of a case of protruding maxillary incisors; the child was fed with a ‘boat-shaped bottle,’ had been free from 'tonsils and adenoids,' had not used a 'comforter,' but had sucked the lip vigorously and, even at the time when the models were taken, was addicted to that habit.

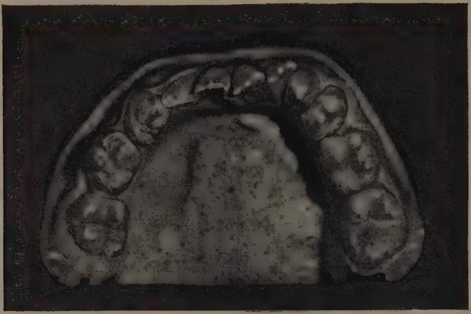

FIG. 325.—View of the mandibular teeth of the case shown in fig. 323.

There can be little doubt that vigorous sucking of the lip is a powerful factor in producing protrusion of the teeth. One has seen a child, three and a half years of age, addicted to this habit, whose mandibular incisors had been driven backwards over the tongue and were placed about three-quarters of an inch behind the maxillary teeth.

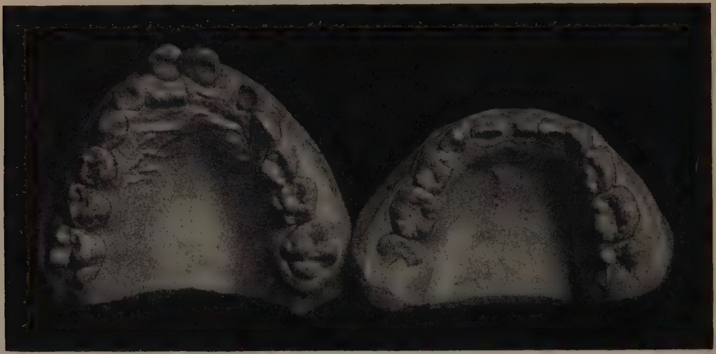

FIG. 326.—A case of superior protrusion due to persistent thumb sucking. Breast-fed. No adenoids.

It is possible that in cases of lip-sucking the backward pressure in the mandible may lead ultimately to changes in the neck of the condyle, a subject referred to on pp. 119 and 120.

The models (figs. 326 and 327) are from a case of persistent thumb-sucking.

Group 2.—There is deficient growth of the premaxilla due to the presence of adenoids, the projecting incisors presenting a crowded arrangement. The specimen (fig. 328) is an example of this type of superior protrusion. The permanent first molars are in normal occlusion ; the canines and premolars have an abnormal forward slope ; the maxillary incisors are crowded together, the apical portions of the roots being directed towards the median line, with the roots of the second incisors partly covered by those of the first incisors. The mandible is better developed than the maxilla. The mandibular incisors and canines are crowded, and present a fan-shaped arrangement which is due to the pressure on the incisors exerted by the growing canines. The incisors are wedge-shaped teeth, the thin ends of the wedges being at the apices. If four wedge-shaped pieces of wood are placed side by side pressure on the thin ends of the wedges will force the centre wedges upwards, and this is exactly what happens with the mandibular incisors when the growing canines exert pressure on the roots of the second incisors (fig. 329).

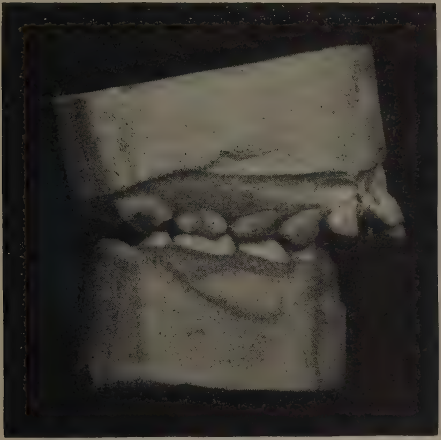

FIG. 327.—Side view of case shown in fig. 326.

In discussing the effect on the jaws and teeth of the presence of adenoids, it was shown that adenoids usually conduce to incomplete growth of the premaxilla, which results in a crowded arrangement of the incisors, also to a narrowing of the arch and, in marked cases, the anterior part of the arch may be pushed forward, causing superior protrusion. It is doubtful, however, whether the extreme protrusion seen in many of these cases is wholly to be attributed to the presence of adenoids and to the effects resulting therefrom. If a considerable number of children suffering from adenoids are examined it will be found that, while

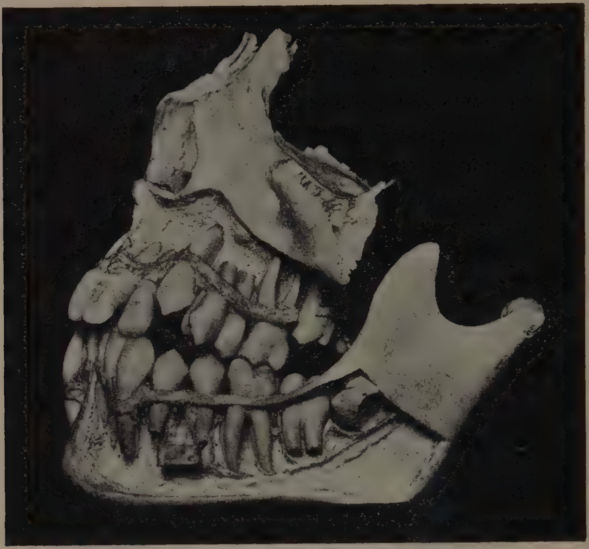

Fig. 328.

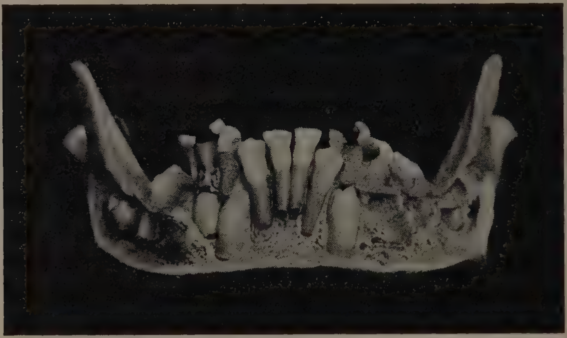

Fig. 329.

many of them have superior protrusion, a greater number will have a crowded condition of the teeth without superior protrusion. The frequency of abnormal occlusion in cases of superior protrusion would suggest that other causes are operating to produce this forward movement of the arch. In the majority of cases included in this group also there is a history of the use of the 'comforter,' or of lip- or thumb-sucking, and these habits may be the determining factor in causing the protrusion. The models (figs. 330 and 331) are from a hand-fed child which suffered from

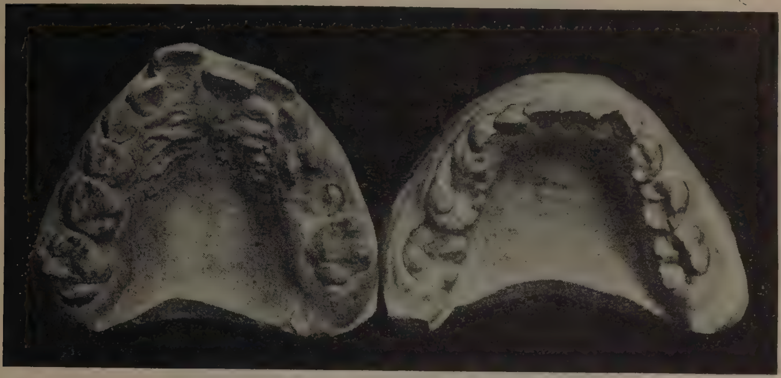

Fig. 330.—Hand-fed with a boat bottle. Suffered from adenoids until eight years of age. Comforter used for nine months. Maxillary incisors show a crowded condition. Mandibular incisors impinge on maxillary gums.

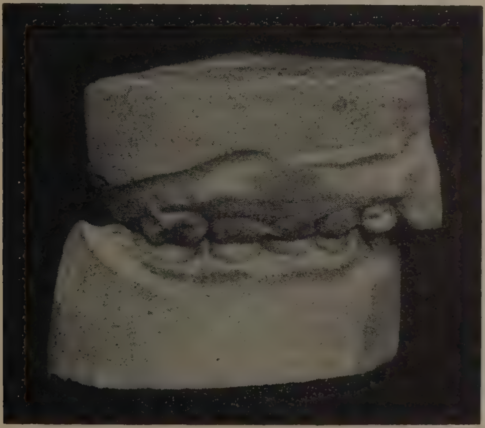

Fig. 331.—Side view of case shown in fig. 330.

adenoids until eight years of age and had used a ‘ comforter ’ for nine months.

A postnormal occlusion is often present in these cases and may be caused in the same manner as in Group 1.

Group 3.—In this group there is deficient growth of the maxilla as a whole, accompanied by lack of growth of the air sinuses and bones of the face. The skull shown in fig. 335 illustrates the morbid anatomy of this group of cases.

The distance between the cutting edges of the maxillary incisors and the labial surfaces of the mandibular teeth is 4 mm., the cutting edges of the mandibular teeth being quite free from the palatal aspects of the maxillary teeth. The occlusion of the permanent first molars is normal.

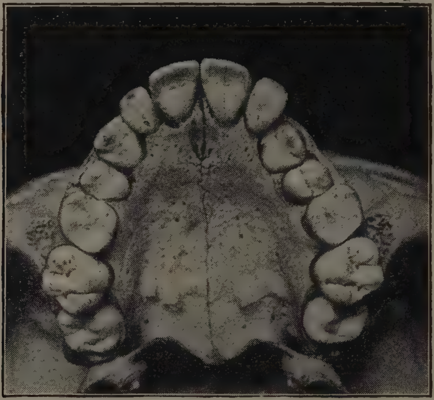

Fig. 332.

The palatal aspect of the maxillary arch is shown in fig. 332, and it will be observed that (1) the incisors and canines slope unduly forwards; (2) the occluding surfaces of the permanent first molars face slightly backwards; (3) behind the erupting second molars there is no development of a tuberosity; (4) there is a narrowing of the arch in all directions, more especially in the region between the canines and first premolars. The widths are as follows: between the (*a*) permanent first molars 34 mm.; (*b*) first premolars 25 mm.; (*c*) canines 20 mm.[1]

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[1] The measurements between the premolars and canines are approximate, as the teeth have not fully erupted.

The arch of the mandibular teeth is regular, and the anterior teeth are somewhat more vertical than in the normal. Side views of the skull (fig. 335) are illustrated in figs. 333 and 334

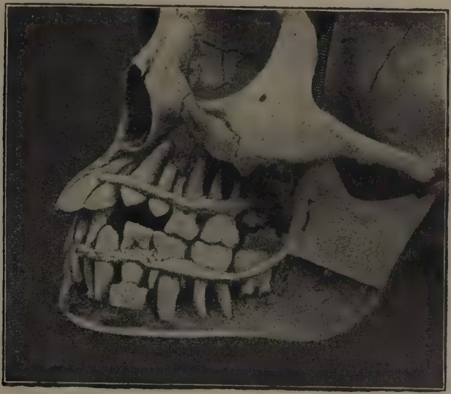

FIG. 333.

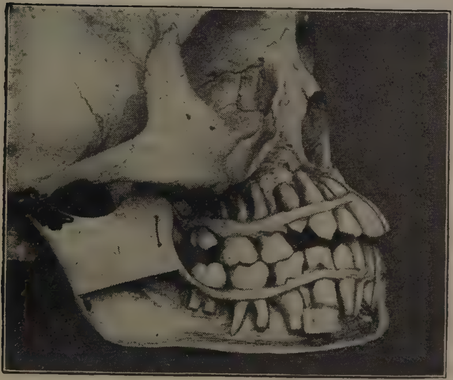

FIG. 334.

and show the abnormal slant of the maxillary incisors. Examined more in detail, it will be noticed that the roots of the incisors converge towards the middle line. The occlusion of the first molars is normal, and they are placed directly under the malar process. The outer alveolar process had been removed when the skull passed into the collection, and it is therefore impossible to define the extent of the sinus, but it would appear from an examination of the specimen that the sinus was very small and did not extend quite to the region of the second premolars. The skull of this child (fig. 335), if compared with the skull of a normal child about the same age (fig. 336), shows a lack of growth of the frontal sinuses and the bones of the face. The comparative measurements of the skulls point to a lack of growth of the maxilla. The effect of this lack of growth would be to narrow the space available for the teeth and cause a crowding together of the roots and a forward movement of the crowns. In other words, if the roots of the teeth could be brought forward and spaced by a growth of bone, the crowns would occupy a broader circle and the teeth would be more upright.

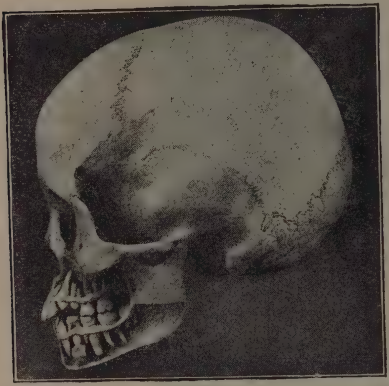

Fig. 335.—Skull of a child about ten years of age, showing superior protrusion.

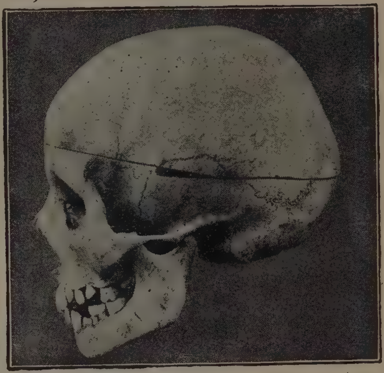

Fig. 336.—Normal skull of a child about ten years of age, for comparison with the skull shown in fig. 335.

Group 4.—In this group there is apparently deficient growth of the mandibular ramus. This causes so close a bite that the premolars and molars are unable to erupt to their normal height, and the result is that the mandibular incisors, being on a higher level, exert undue pressure in a forward direction on the backs of the maxillary incisors, on which they act as on an inclined plane, and so force them into undue prominence. The mandibular incisors are also spaced and inclined forwards.

Apart from the foregoing, the arches are well formed and of normal width.

Group 5.—The greater part of the maxillary teeth occlude external to the mandibular teeth (see fig. 337). The pathology of these cases is obscure. The irregularity may be due to over-development of the maxilla or under-development of the mandible.

In cases of superior protrusion the deformity increases as the child approaches the age of twelve years. This is due to the pressure from the erupting canines.

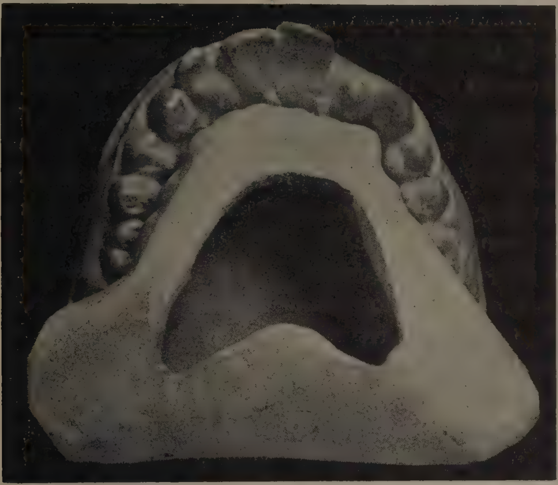

FIG. 337. (See also figs. 123 and 124.)

Treatment.

In all cases it is of prime importance that habits such as finger- or lip-sucking must be discontinued. This is usually a simple affair, except with regard to sucking the lower lip, which is a habit often persisted with till about the age of puberty, and not an easy one to break ; the surest method of ensuring its discontinuance is that which is brought about by treatment approximating the maxillary and mandibular incisors to such an extent that the lip cannot be accommodated between the closed teeth. It is of equal importance as a preliminary to any form of treatment to ensure nasal patency and functioning when this is not present.

Nasal patency being present, the use of **oral screens** is often of considerable value by doing away with the habit of 'mouth breathing,' and so preventing the anterior salivary stasis and consequent gingivitis which almost invariably accompany it, and at the same time keeping the lower lip or tongue (usually the former) from finding accommodation between the closed resting teeth. Their value lies in their use during sleep when the patient's volition is in abeyance.

According to H. O. Dickin,[1] who has had a wide experience with oral screens: 'During normal nasal respiration a negative intranasal pressure occurs on inspiration and a positive on expiration; the passages warm the air and add moisture as required; a varying negative pressure persists in the mouth.

'In mouth-breathers it is necessary to re-establish and maintain normal conditions, particularly during sleep, and this is best done by means of oral screens. It is found that this means of restoring normal function tends automatically to reduce many forms of dental irregularity.

'By this means the widths of the maxillary and mandibular arches may be increased considerably in comparatively short periods; the confinement of the tongue within the arches and the lower lip outside, tends to restore inter-arch harmony and the normal occlusal relationship of the teeth (figs. 338 and 339).

'Owing to the range of variation in the age at which growth ceases no hard and fast rules can be laid down as to when such treatment may not be given to influence dental irregularities, but treatment should always be given as early as possible (and may be started even before the commencement of the third year), as improved facial development will be more readily influenced during the growth period, and may even regain normality before growth is completed. In this manner a wider U-shaped arch and a flatter palate are produced and a dental occlusion approximating, if not actually normal.

'Initially cases requiring oral screen treatment show contracted arches and the partial nasal stagnation predisposes to further infection; this contraction of the arches tends to increase, so producing malocclusion, protrusion of the anterior teeth and a varying degree of facial distortion accompanied by gingivitis.

'In designing an oral screen articulated models showing the full depth of the sulci are necessary, with the bite opened or closed as conditions may require; these are suitably padded with soft metal and wax on the labial and buccal aspects as far back as the distal aspects of the permanent first molars and as deep into the sulci as may comfortably be borne. Dies and counterdies are then made from a plaster impression and model of the planned screen. Aluminium flat of not less than 99·5 per cent. purity and ·04″ gauge, rolled as hard as possible, is swaged whilst protected by suitable paper from contamination by the dies, as traces of metallic contamination may give rise to appreciable galvanic action and cause salivation.

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[1] See also *Proc. Roy. Soc. Med.* (Odonto. Sec.), 1934, xxvii, 51.

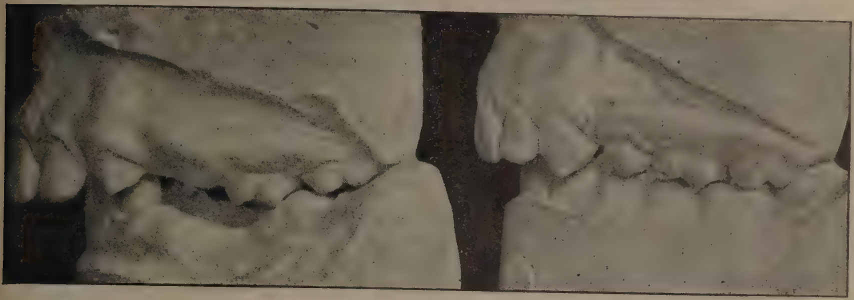

Fig. 338.—Models showing the change brought about solely by the use of an oral screen inserted at age 10 years 6 months. (H. O. Dickin.) (From *Proc. Roy. Soc. Med.*)

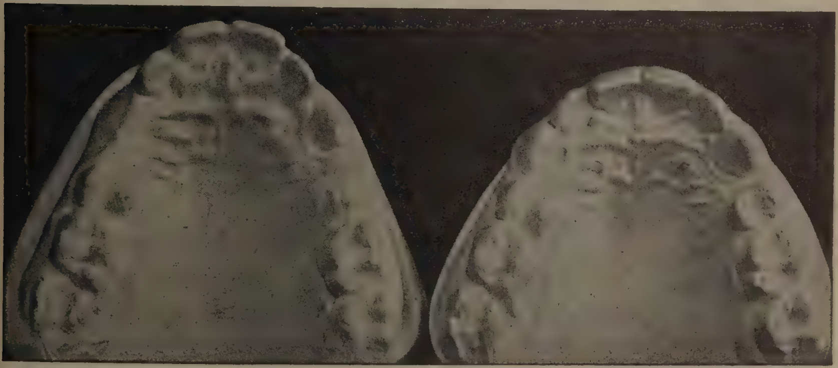

Fig. 339.—Models showing change in the maxillary arch in the case illustrated in fig. 338. (H. O. Dickin.) (From *Proc. Roy. Soc. Med.*)

‘A well designed screen for a young patient may become unsuitable and even unusable in a few weeks owing to the changes which it has already brought about, so that a new screen is required.

'Serial models recording progress are highly desirable, indeed a necessity, and the same remarks apply to radiograms in profile as recording development of facial structure. For the latter a fixed target film distance of one metre gives reasonable perspective, the centric ray should be directed at right angles to the sagittal plane to a point at the posterior limit of the nostril. Two whole plate films may be placed in one cassette, the front film unscreened and the back film between intensifying screens ; this results in the soft tissues appearing on one film and the deep structures on the other.'

Another aid in the treatment of this condition is by means of a lip exerciser ; the exercise is to train those who habitually hold the lips apart to keep them closed, and consists of a flattened piece of stout aluminium wire about three inches long with a hole bored at one end for attachment by a thread to the child's clothing to prevent accidental swallowing. The lips should be dry, and the exerciser held lightly with, and parallel to, the lips only ; there should be no puckers in the chin when it is held ; these often occur at first but should disappear after a week's use.

When out walking or on other occasions when the exerciser is not available a portion of a match-stick with the head off, or other suitable object may be used, though such object is not essential if the patient will keep the lips closed without it.

The exercise should be done at least twice a day, morning and evening ; also at other times : the more it is practised the sooner will the result be obtained. The length of time the exerciser is held continuously at the outset varies considerably from a few seconds to as many minutes : its use should cease before tiredness ensues. The exerciser should be held longer each day, also the length of time it is held continuously should be increased each day. After some weeks' practice it will be possible to hold the exerciser continuously for half an hour : to hold it for an hour without dropping it should be the aim. The time when the exercise is performed should be devoted to some other occupation, *e.g.* reading, because the object is to keep the lips closed without conscious effort ; this may not be possible at the outset, but should be in a short time.

Regularity and perseverance are essential for good results : these depend entirely on the child, with encouragement from those in charge.

Never hold the lips forcibly together—gentle contact is all that is required.

The use of this appliance in conjunction with the oral screen appears to be of value, as the one can but assist the other.

When the jaws are well formed, the occlusion of the cheek teeth normal, and the maxillary incisors well spaced, the only treatment required is the retraction of the maxillary incisors, accompanied sometimes by the protrusion of the mandibular incisors and retention in the corrected position.

In all other cases it is advisable to make room in the arch by the extraction of teeth before commencing to retract the incisors. For the purpose of describing the treatment of these cases it will be convenient to divide them into two groups, namely, those in which the mandibular incisors do not impinge upon the cingula of the maxillary incisors or upon the gum posterior to those teeth, and those in which they do impinge on one or the other.

As a preliminary to treatment, the jaws should be radiographed to ascertain (1) the position of the canines in relation to the first premolars and the second incisors, and (2) the presence or absence of the second premolars.

(i) Cases in which the Mandibular Incisors do not impinge on the Cingula of the Maxillary Incisors.—(*a*) *Where the permanent first molars are savable.* The method of treatment will depend upon the presence or absence of the second premolars and the relation of the canines to the second incisors. We will first consider the **cases where the second premolars are present and the canines are in a position favourable for being moved backwards.** In discussing protrusion of the maxillary teeth, attention has already been drawn to the following facts :—

(α) The condition is often accompanied by a certain degree of abnormal growth of the bone, which disturbs the normal relation between the jaws and the teeth.

(β) The maxillary, and not the mandibular, arch is at fault in the majority of cases.

(γ) The protrusion increases rapidly with the eruption of the canines.

These facts suggest that extraction of teeth to relieve the crowding, and extraction at an early age, is the best treatment. Treatment by early extraction gives the most permanent and satisfactory results. Nevertheless, it is not universally supported, and many practitioners deprecate any attempt at correction until the permanent second molars and the canines have erupted. It is not easy to discover any advantage that will accrue by postponing treatment, while the disadvantages of delay are obvious, namely :—

(1) The canine in erupting will move forward and accentuate both the protrusion of the incisors and the crowding of their roots.

(2) When treatment is commenced, the canine must be retracted by mechanical means, and in this process, instead of moving bodily back, and so giving true relief to the pressure in the front of the mouth, the crown only will swing backwards, leaving the apical end of the root in much the same position as it erupted. The roots of the anterior teeth will thus remain more or less crowded, and there will consequently be a constant tendency for the crowns to relapse into their former position.

(3) The retraction of the incisors will later on take a longer time, partly because the protrusion will have become more pronounced, and partly because the alveolar process will be more resistant.

(4) Owing to the prolonged mechanical treatment which will become necessary, disorganisation of molar and premolar occlusion is more likely to ensue.

With treatment carried out before the eruption of the canines, these teeth will erupt well back, pressure will immediately be taken off the incisors, the protrusion will not increase, and the mechanical treatment necessary will be slight. Still further, the teeth will come into good alignment, and the tendency to relapse will be reduced to a minimum. If early treatment is adopted, adequate space for the canines should be obtained by removing the unerupted first premolars as soon as the canines show signs of erupting. The method of carrying out this operation is described in Chapter XXVII.

After the removal of the premolars, the case should be left alone until the canines have fully erupted, but the patient should be strongly urged to keep the lower lip in front of the maxillary teeth. Occasionally, this simple expedient is sufficient to bring about a complete cure of the case. The effect of removing the pressure of the canines on the incisors is interesting. The second incisors soon show a tendency to travel backwards, and this movement indicates a complete relief of the crowding of the anterior teeth. The incisors are eventually brought in with a simple mechanical contrivance, the teeth being retained in position until after the permanent second molars have erupted.

With regard to the mandibular teeth, the treatment must depend on the condition existing. If they are crowded, the first premolars must be removed, but their removal should be delayed until the canines have commenced to erupt. Little advantage is gained by removing the mandibular premolars at an early age, and in this respect the treatment differs from that recommended in the case of the maxilla.

When superior protrusion is treated at an early age excellent results are obtained ; there is no tendency to recur ; the amount of mechanical treatment necessary is trifling, and the occlusion of the premolars and molars is good. The cases shown in figs. 340 to 348 are examples of this mode of treatment.

In cases where the *roots of the permanent canines are placed towards the middle line or lie anterior to the roots of the permanent second incisors*, the best plan is to extract the permanent second incisors and the deciduous canines before the permanent canines show signs of erupting. The case should then be left alone until the canines have fully erupted, when the first incisors can be retracted. The objection to the removal of permanent second incisors was discussed on p. 220 *et seq*.

If the second premolars are missing it is advisable to extract the deciduous first and second molars as early as possible, and then insert an appliance to prevent the permanent first molars from moving forward. The removal of the deciduous molars will allow the first premolars and permanent canines to move backwards and so relieve the pressure on the anterior part of the arch. The retraction of the incisors should be delayed until the permanent canines have fully erupted.

If the permanent second incisors are missing the deciduous second incisors and canines should be extracted at an early age, and when the permanent canines have fully erupted the first incisors should be retracted. If the permanent second incisors are missing and there is risk of spaces remaining between the permanent canines and the posterior teeth, it may be advisable to extract the deciduous molars and so permit the permanent first molars to move forward and assist in closing the spaces.

(*b*) *When the permanent first molars are unsavable.*—The extraction of the maxillary teeth may be undertaken as soon as the permanent second molars are sufficiently through to allow of their retention in position by means of a splint plate (fig. 249). This plate, as before mentioned, will prevent the permanent second molars from moving forward, and will allow them to erupt fully. It will also permit the premolars to travel back, provided that the bite of the mandibular incisors on the plate

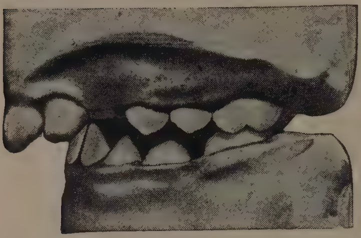

FIG. 340.—Before treatment.

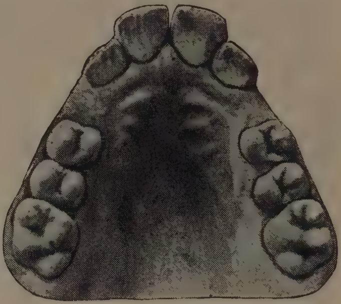

FIG. 341.—Before treatment.

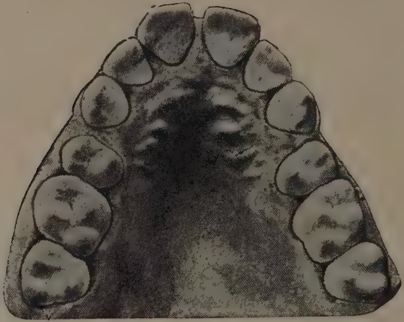

FIG. 342.—Stage at which mechanical retraction of the teeth was commenced.

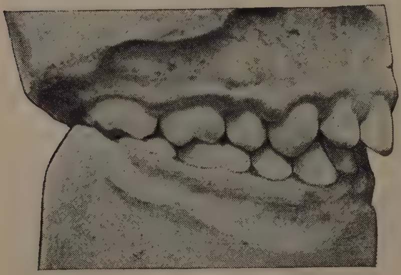

FIG. 343.

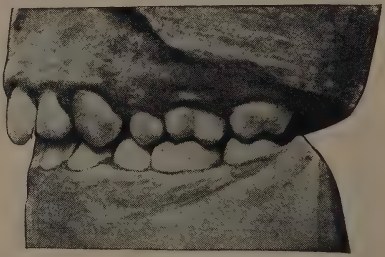

FIG. 344.

Case shown in figs. 340 and 341. After treatment.

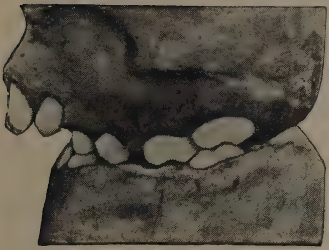

FIG. 345.

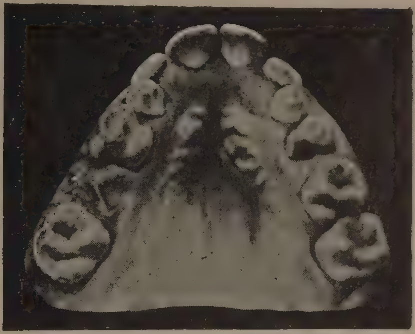

FIG. 346.

Before treatment.

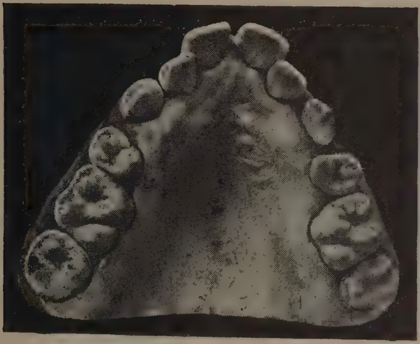

FIG. 347.

Stage at which mechanical retraction of the teeth was commenced.

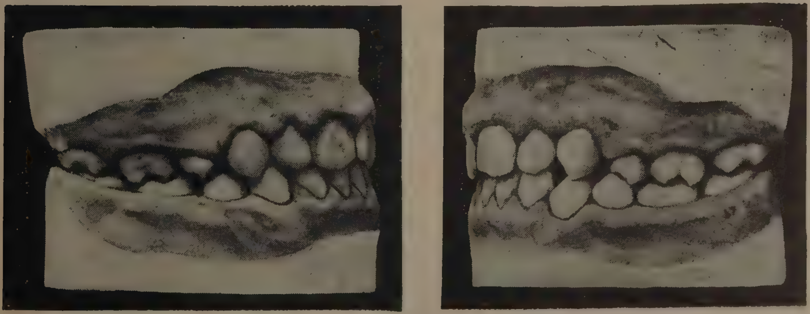

FIG. 348.

Case shown in figs. 345 and 346. After treatment.

is so arranged that the maxillary and mandibular premolars are separated from one another during occlusion. No attempt should be made to move back the premolars by mechanical means until the permanent second molars are firmly implanted, as the molars are liable to move forward if mechanical force is used too soon.

For retracting the premolars, a plate similar to that shown in fig. 349 may be used. It is important that the plate should occlude correctly with the mandibular teeth in order that its rigidity may be augmented. It is advisable to retract only one premolar on each side at a time. If both premolars are retracted simultaneously, the second molar is very likely to move forward, as the resistance offered by two premolars is frequently greater than that of one molar, especially if the latter is not fully erupted. The premolars should be moved far enough back to allow the anterior planes of the maxillary teeth to strike the posterior planes of the mandibular teeth, otherwise the posterior planes of the maxillary teeth will tend to drive the mandibular teeth backwards and so cause the postnormal articulation to persist (see figs. 350 to 352). If possible, the mandibular permanent first molars should be retained until the maxillary premolars have been brought into correct occlusion. When the premolars are sufficiently retracted, the canines and then the incisors may be treated.

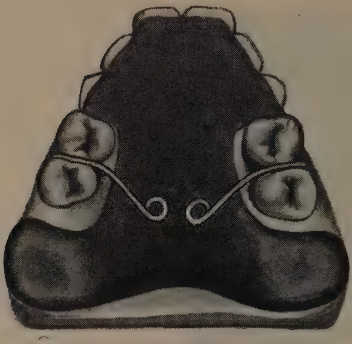

FIG. 349.

The results obtained are not very satisfactory, as there is usually a tendency to relapse, due in a measure to the fact that extraction of the permanent first molars gives but little relief to the crowding of the anterior teeth.

With excessive protrusion and unsavable permanent first molars, it may be necessary to remove the first premolars, in addition to the permanent first molars. The permanent first molars must be removed because they are unsavable, but their removal will not materially aid in the treatment of the protrusion. If the protrusion is left alone, the teeth are almost certain to be lost at an early age from parodontal disease. If the protrusion is to be treated with any hope of obtaining a permanent result, the premolars must be removed, and, when the one alternative is weighed against the other, the advantage would seem to be in favour of the extraction of the premolars and the retention of the front teeth.

When the permanent first molars are unsavable and the permanent second incisors are absent or the permanent canines are placed in an unfavourable position for moving backwards, the extraction of the permanent first molars should be carried out before the eruption of the permanent second molars in order that these teeth may move forward in contact with the second premolars. The remainder of the treatment should follow the lines suggested on p. 259.

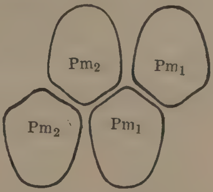

FIG. 350.—Diagram of articulation of superior protrusion cases which often require retraction of the maxillary premolars.

In cases of superior protrusion which are not seen until after the permanent canines have erupted, a prolonged spell of treatment is usually necessary, and the results obtained are often far from satisfactory.

In the foregoing account, treatment has been based on the principle of creating a space on either side of the arch and bringing the front segment backwards. Some practitioners maintain that cases of protrusion can best be treated by '*jumping the bite*,' and base their treatment on the assumption that the position of the maxillary teeth is normal and that the trouble

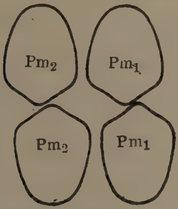

FIG. 351.—Diagram to illustrate the least extent to which maxillary teeth must be retracted. The posterior plane of the mandibular teeth will tend to drive the maxillary teeth backwards.

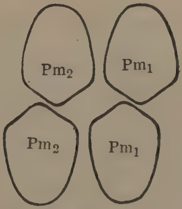

FIG. 352.—Diagram to illustrate insufficient retraction of maxillary teeth. The posterior planes of the maxillary teeth will tend to drive the mandibular teeth backwards and the abnormal articulation of the premolars will persist.

is due to a retraction of the mandible. For example, in the case fig. 353 the mandibular premolars and molars are one cusp unit behind the correct occlusion. If the patient can be made to acquire the permanent habit of bringing the mandible forward so as to make the teeth articulate normally, the bite will have been 'jumped.' The subject is one which has been voluminously written upon, and on which there is much diversity of opinion. The means usually employed is to adapt to the maxilla a plate at the anterior part of which is an inclined plane shaped to bring the mandible forward in the act of closing the mouth.

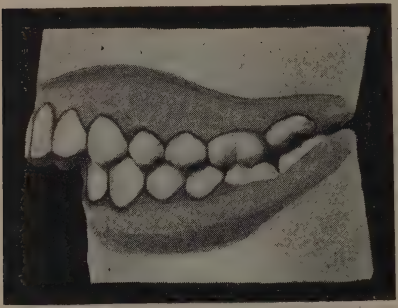

FIG. 353.

It is clear from the cases recorded that patients can be made to acquire, at least temporarily, the habit of protruding the jaw, and thus 'jumping the bite.' Does this habit become permanent, and, if so, what structural changes take place in the parts involved? By some[1] it is maintained that changes take place in the glenoid cavity (1) by an extension of the condyle of the mandible, which practically amounts to a bending backwards of the neck; (2) by the filling up of the cavity posterior to the condyle. These explanations do not seem compatible with our knowledge of the anatomy and physiology of the temporo-mandibular articulation. A more likely explanation is that the teeth move forward in the sockets. When the teeth are occluded in the new position, namely, with the bite jumped, the muscles must constantly tend to retract the mandible to its old position; this backward tendency is arrested by the maxillary teeth, with the result that the lower teeth are drawn forward in their sockets and absorption and re-disposition of bone in the tooth sockets take place as when a tooth is moved by mechanical means.

It is possible, and doubtless takes place in some cases, that the ever present tendency of the mandible with its teeth to move backwards into its old position, produces a compensatory movement of the maxillary teeth by means of the occlusal planes of the individual mandibular teeth acting on those of the maxillary teeth.

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[1] See *Trans. World's Columbian Dental Congress*, ii, 760.

E. H. Angle,[1] in referring to this point, states that cases examined four years after the termination of treatment show the backward tilt of the maxillary teeth and the forward tilt of the mandibular ones.

Some practitioners treat cases similar to that shown in fig. 353 by means of intermaxillary traction. Expansion arches are adjusted to both jaws, and the teeth are spread to the desired position; the expansion arches are then connected by elastic bands, as shown in fig. 354. It is claimed that by these means the mandibular teeth can be easily moved forwards and the maxillary backwards. When the teeth are in normal occlusion, a retention apparatus is applied. The models of a case treated in this way are shown in figs. 355 and 356. A careful examination of the models shows that although the premolars were brought into correct occlusion the permanent second molars have been rendered almost functionless.

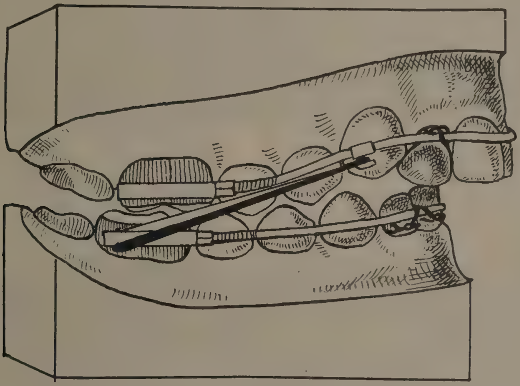

FIG. 354.

Where the protrusion is complicated by tilting of the permanent second incisors it is difficult to obtain permanent results. The crowns of the incisors are arranged in a fair arch, but in such a way that the roots of the permanent second incisors lie towards the median line. This condition is probably the result of adenoids, or some other form of nasal obstruction. The anterior nares, being functionless, do not develop properly, and so indirectly, and perhaps directly, affect the growth of the premaxillæ. In these cases the permanent canines come down in such a way that their roots lie towards the median line. Retractive measures will draw back the crown, but the pressure

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[1] 'The Treatment of Mal-occlusion of the Teeth.' Seventh Edition.

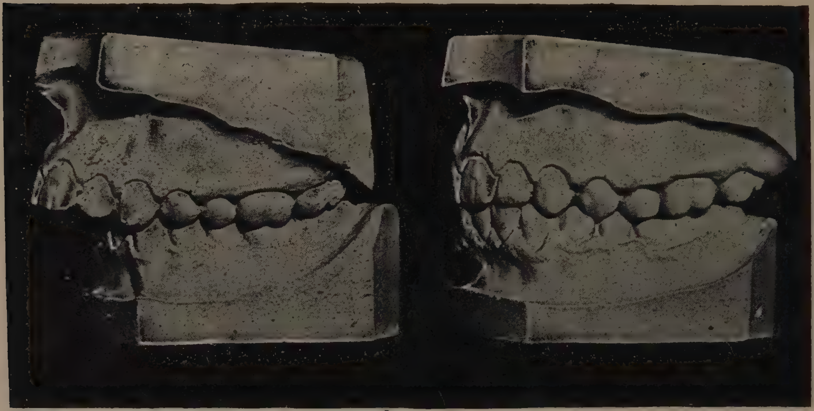

FIG. 355. (From 'Operative Dentistry,' C. N. Johnson.)
Left side.
Before treatment. After treatment.

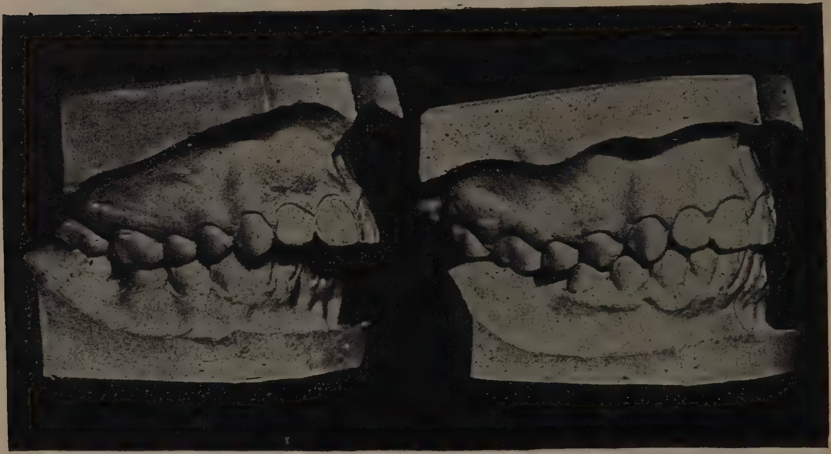

FIG. 356. (From 'Operative Dentistry,' C. N. Johnson.)
Right side.
Before treatment. After treatment.

on the root of the permanent second incisor is not relieved, indeed, may even be increased. The space gained in the arch by the retraction of the permanent canines will admit of retraction of the incisors to a certain extent, but the effect of retraction of the incisors is that their roots will become still more crowded, and there will be a strong tendency to relapse unless a retention plate is regularly worn at night. The choice lies between (1) leaving the case alone, with the inevitable result that the condition becomes worse and parodontal disease supervenes; and (2) treating the case, and, after the teeth are retracted, impressing on the patient the necessity of wearing a retention plate at night.

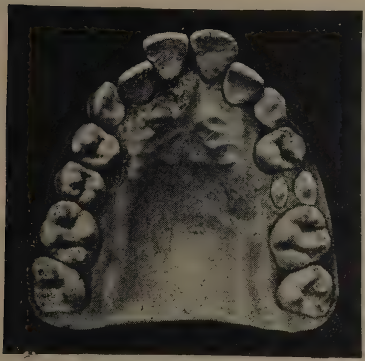

Fig. 357.[1]

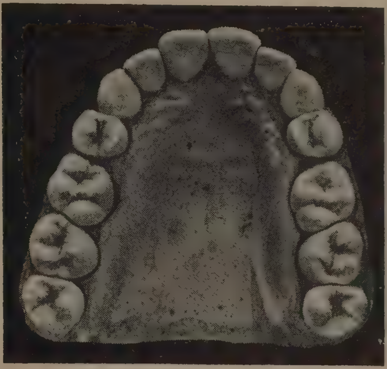

Fig. 358.
Case shown in fig. 357 after treatment.

In the case shown in figs. 357 and 358 the second incisors have been forced slightly behind the first incisors. The first premolars were removed and the anterior teeth retracted. At the age of twenty-five (fig. 358) there was still a tendency for the teeth to relapse to their former positions unless the retention apparatus was worn at night.

Where there has been considerable interference with the growth of the premaxillæ so that the root of the permanent second incisor is covered by that of the permanent canine, by far the most satisfactory results are obtained by the extraction of the permanent second incisors. A typical case is shown in fig. 359.

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[1] The patient was hand-fed and suffered from adenoids which had not been treated.

(ii) Where the Mandibular Incisors impinge on the Cingula of the Maxillary Incisors, or on the Gum Posterior to these Teeth.—These cases are difficult to treat and the results obtained are frequently unsatisfactory. If the mouth of a patient presenting this form of superior protrusion be examined, it will be noticed that the mandibular incisors are on a higher level than the premolars and molars. This may be due to some abnormal condition of the incisors, the height of the premolars and molars being normal; or the incisors may be normal in their arrangement and the premolars and molars unduly short; or again, both incisors, premolars, and molars may be at fault. In order *that treatment may be successful, it is essential that the mandibular incisors should not impinge on the cingula of the maxillary incisors when the latter have been retracted.* It is therefore needful that attention should be directed to the treatment of the mandibular teeth. It has already been pointed out that the abnormal uprising of the mandibular incisors is due to crowding from lateral pressure of the canines, and that the shortness of the range of the molars and premolars probably arises from a defect in the development of the ramus of the mandible. As a first step in treatment, most practitioners endeavour to raise the bite—in other words, induce the molars and premolars to elongate and so prevent the mandibular incisors from impinging upon the cingula of the maxillary teeth. There are two strong *objections to treatment by raising the bite*: (1) the obstinacy, at times, on the part of the molars and premolars to rise; and (2) their liability to relapse by being forced down again into their sockets. It is not surprising that disappointment attends the practice of 'bite raising,' since the treatment is not in accord with our anatomical knowledge of the condition. *Failure to raise the bite* seems more likely to occur

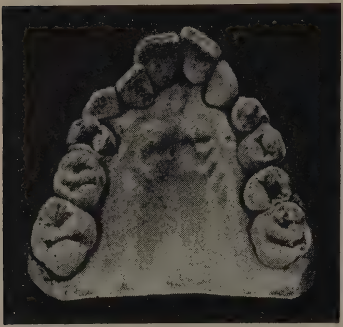

FIG. 359.[1]

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[1] The patient was hand-fed, a tube bottle being used; she had also suffered from adenoids. The adenoids were probably responsible for the interference with the growth of the premaxillæ, and the marked narrowing of the arch was probably due to the use of the tube bottle.

in cases where the molar and premolar region is well developed. There is a common impression that if teeth are separated from one another they will always elongate. That this is not always so will readily be seen by a study of that type of irregularity called 'open bite.' When the ramus is short, the premolars and molars are probably prevented from rising to their normal height. If, therefore, the jaws are separated, the teeth will no doubt rise. It is in these cases that the liability to relapse occurs. Raising the bite does not in any way affect the ramus, which is probably the real cause of trouble. The mouth is in reality propped open artificially and the muscles which close the mandible are in a state of tension. As soon as the plate is removed the muscles have again free play, and the pressure they exert drives back the premolars and molars to their original positions.

In a few cases, with the eruption of the permanent second molars, the bite becomes raised by natural means, probably from a rapid growth of the ramus about this period, and it is possible that where the bite has been raised by artificial methods the same natural causes have contributed to the results obtained.

More reliable results can be obtained by directing treatment to the mandibular incisors. Where the incisors and canines are not very crowded, their cutting edges should be stoned down until they are clear of the maxillary teeth ; at the same time it is most important that all lateral pressure should be relieved by the removal of a premolar or molar. If this is not done, there is a risk that when the third molars erupt they will exert a forward pressure and cause crowding of the anterior teeth. Where the incisors are very crowded, with the permanent canines lying slightly anterior to the permanent second incisors, or where the fan-shaped arrangement is well marked, it is an excellent plan to remove an incisor. The crowding of the remaining teeth is efficiently relieved, and the incisors usually fall to their normal level and are so prevented from impinging upon the maxillary gums or teeth. Removal of an incisor slightly narrows the mandibular arch, and this is a distinct gain as far as treatment is concerned. Some, however, maintain that the narrowing of the arch may be detrimental to the facial expression because it will allow the lower lip to fall in. This view is incorrect. The prominence of the chin is governed by the mental process and is in no way dependent upon the alveolar portion of the jaw, which is alone involved in the removal of an incisor. When the maxillary teeth have been retracted the lower lip should close outside them and should not be dependent upon the arch of the mandibular incisors for its position. A case treated in this manner is shown in figs. 360 to 365. The amount of protrusion is shown in fig. 360.

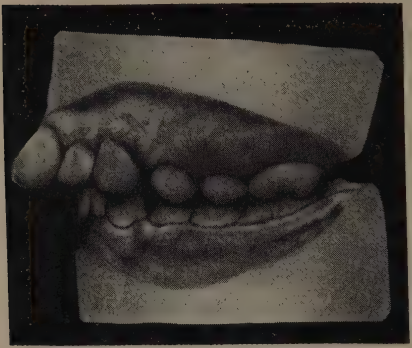

Fig. 360.—Left side before treatment.

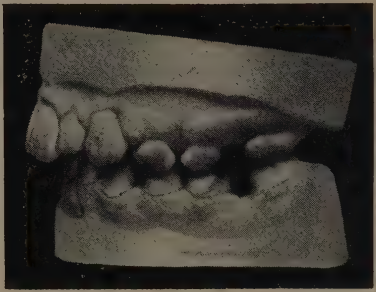

Fig. 361.—Left side after treatment.

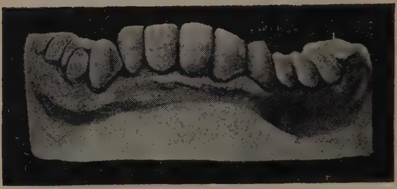

Fig. 362.
Mandibular incisors before treatment.

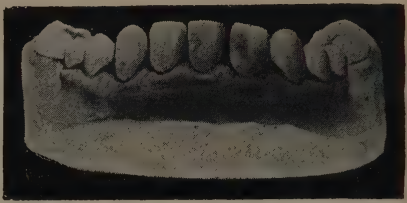

Fig. 363.
Mandibular incisors after treatment.

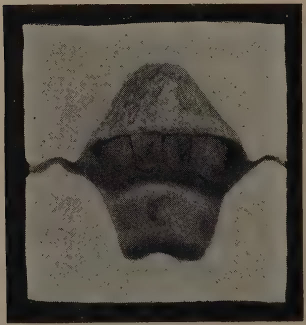

Fig. 364.—Showing relation of the mandibular incisors to the maxillary teeth at the commencement of treatment.

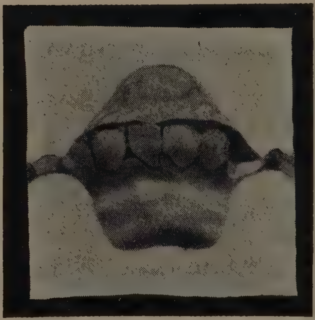

Fig. 365.—Showing relation of mandibular to maxillary incisors after treatment.

An examination of the models shows that as regards occlusion with the mandibular teeth the maxillary right premolars are erupting normally; on the left the maxillary premolars are not quite half a tooth in advance of their correct positions, the anterior planes of the maxillary teeth just striking the posterior planes of the mandibular teeth. The mandibular incisors are crowded and show the typical heaped-up appearance already alluded to (fig. 362). The cutting edges of these mandibular incisors strike the gum just posterior to the maxillary incisors (fig. 364). The direction of the roots of the mandibular incisor teeth indicated that the left first incisor should be removed. This tooth having been extracted, the case was left for a year, as the permanent first molars, which were unsavable, had to be removed, and this could not be done when the patient was first seen, as the maxillary permanent second molars had not then erupted. When examined one year afterwards, the gap caused by the removal of the mandibular first incisor had closed up, the teeth had apparently dropped down to a lower level (see fig. 363), and were quite free from the maxillary gum. By the removal, therefore, of this first incisor one of the great difficulties in treating this type of case had been overcome. The permanent first molars were now extracted and a splint plate put in to keep the permanent second molars from moving forward and at the same time allow the premolars to travel back. The permanent canines were then retracted and the incisors brought in. The result is seen in figs. 361 and 365. The mandibular incisors are quite clear of the maxillary teeth and the lower lip also passes in front of them. The maxillary teeth could have been retracted still more, but this was not considered advisable for æsthetic reasons. The case has shown no tendency to relapse.

The question whether, after treatment, cases of superior protrusion tend to relapse has long been the subject of much controversy. It may, however, be safely predicted that there will be no tendency to relapse if we can—

(1) Ensure that the lower lip in occlusion passes in front of the maxillary incisors.

(2) Ensure that the mandibular incisors do not press unduly upon the backs of the maxillary incisors.

(3) Prevent crowding of the maxillary incisors and canines.

The tendency to relapse will be in proportion to the failure to overcome these difficulties.

(B) INFERIOR PROTRUSION—UNDERHUNG BITE.

The nomenclature of this condition is in a state very similar to that of superior protrusion, in that as some cases are due rather to deficient development of the maxilla or premaxilla, or both, they may equally well be called superior retrusion, and the condition present should in each case be named according to where the fault is deemed to lie.

In this condition, when the mouth is closed, the anterior maxillary teeth pass within the arch of the mandibular teeth, instead of outside it; in other words, the mandible protrudes. This abnormal arrangement of the teeth may be limited to the incisors and canines, or may involve the premolars and molars as well. Protrusion of the mandible is a natural condition in the edentulous.

Etiology and Pathology.—In the more simple examples of inferior protrusion the deformity is due to a faulty occlusion of the teeth in jaws otherwise well developed; but in other cases it arises from faulty growth of the bones themselves.

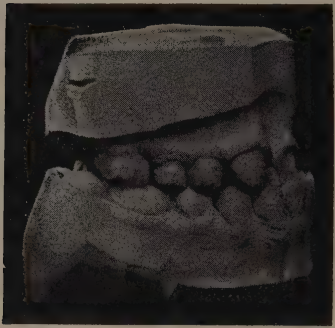

Fig. 366. (From 'Text-book of Operative Dentistry' (Kirk).)

In the first group may be included those examples which are traceable to *habits*, one of which is *the constant protrusion of the mandible* and another the *hooking of the fingers over the mandibular teeth*, causing a forward movement of the incisors until they occlude anterior to the maxillary teeth.

In some cases the deformity is due to the *eruption of the maxillary permanent incisors internal to the existing deciduous teeth.* The permanent incisors, when fully erupted, pass behind the mandibular incisors and the latter assume a more forward position. The maxillary permanent canines in erupting move slightly towards the median line and lock the incisors in their malposition. Cases of this character are recognised by the general direction of the incisors, the maxillary teeth sloping backward and the mandibular teeth forward. The occlusion of the premolars and molars is usually normal. In fig. 366 is shown an example of this type of protrusion.

In the second group the etiology and pathology are frequently obscure. In many cases the trouble can be traced to a *defective growth of the maxilla* associated possibly with adenoids. The lack of growth may be limited to the premaxillæ, so that the only teeth involved in the deformity are the incisors, or the maxillæ may be generally affected. An examination of the mandible will not disclose any defect of growth.

In other cases the *fault is traceable to the mandible.* In some examples the mandible seems to be unduly large, the relation of the body of the bone to the ramus being normal. In others the protrusion appears to arise from a thrusting forward of the body of the bone due to an excessive growth of the ramus. In the skulls shown in fig. 367 this abnormality is well shown. It will be noted that in the skull marked (A) the mental process is unduly prominent, a condition not infrequently seen in these cases. An examination of the skulls will also show that the mandibular condyle articulates correctly in the glenoid cavity.

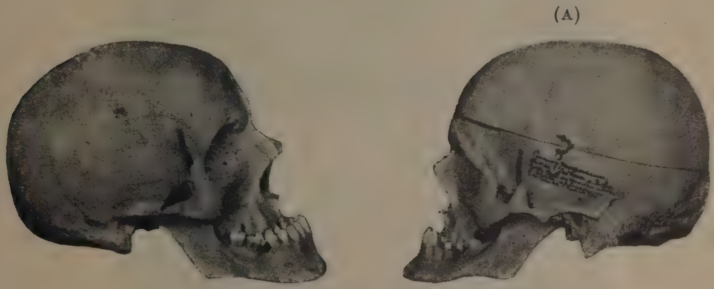

Fig. 367.

This variety of inferior protrusion is occasionally transmitted through generations, a classical example being seen in the Hapsburg royal line.

Fig. 368 shows the models of a case of inferior protrusion due to faulty growth of the mandible. The mandibular teeth in occlusion are in advance of the normal. Fig. 369 represents the models of a boy aged five years, showing well-marked protrusion of the mandible.

In some cases of forward bite, however, the articulation of the condyle is not normal, and in fig. 370 the condyle rests on the eminentia articularis. M. Cryer,[1] who drew attention to this peculiarity, inclines to the view that the abnormal position of the condyle in these cases may be due to the use of forceps at birth ; the mandible, he thinks, may have been drawn forward, and, if the displacement is not subsequently reduced, the temporo-

[1] *Dental Cosmos*, November 1906.

mandibular articulation will be changed from the glenoid fossa to such position as the condyles may assume.

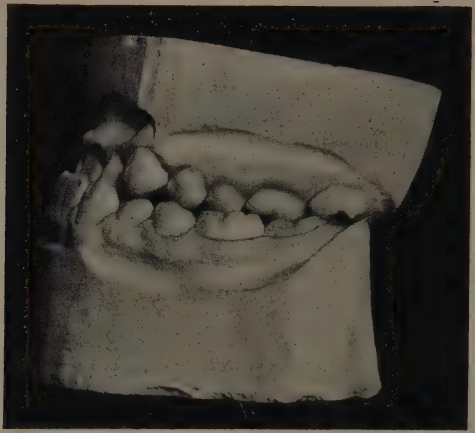

Fig. 368.

L. S. Chilcott [1] thinks that similar injury may follow difficult 'breech presentation' cases. Cryer also quotes a case of forward bite associated with malposition of the condyles in a man aged thirty-five, who had been a case of breech presentation.

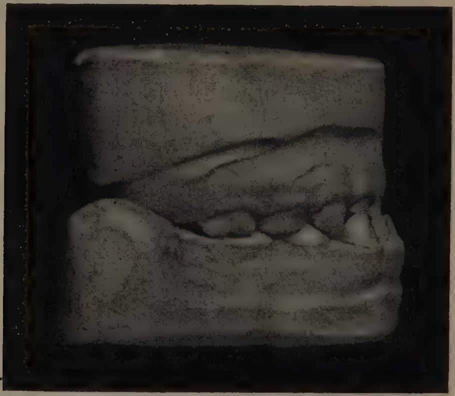

Fig. 369.

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[1] *Dental Cosmos*, March 1906.

In acromegaly the mandible is frequently found enlarged.

In examining patients showing inferior protrusion, it will often be noticed that when the jaws are in repose the maxillary and mandibular incisors are in contact, leaving a space between the opposing premolars and molars, but during mastication the mandible is brought forward to obtain the necessary occlusion of the posterior teeth. This point should be remembered when cases are being examined with the object of ascertaining how far back the mandible can be taken.

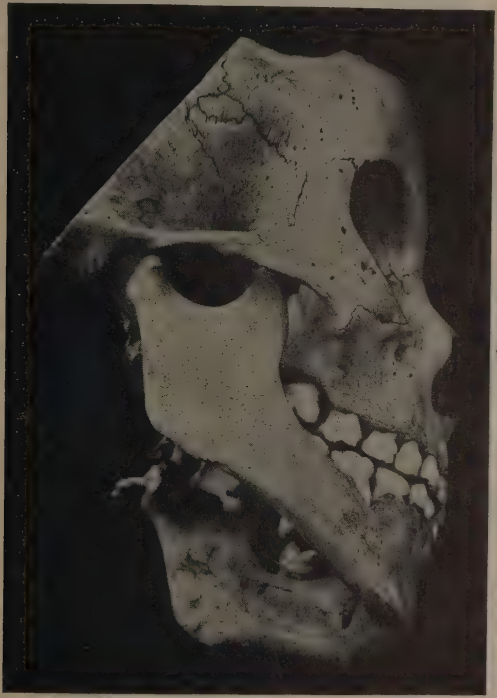

Fig. 370.

Treatment.

In a few cases the condition is cured by natural means ; this happened in the boy whose models are shown in fig. 369. No treatment was adopted, but by the age of sixteen he presented the condition shown in fig. 371.

Where the irregularity is limited to the dental arches the mandibular incisors may be brought into correct position by mechanical means. A plate capping the mandibular premolars and molars, with a half-round gold wire impinging on the labial surfaces of the protruding teeth, will usually suffice.

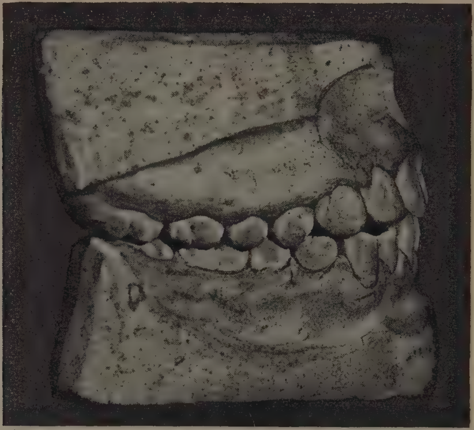

Fig. 371.

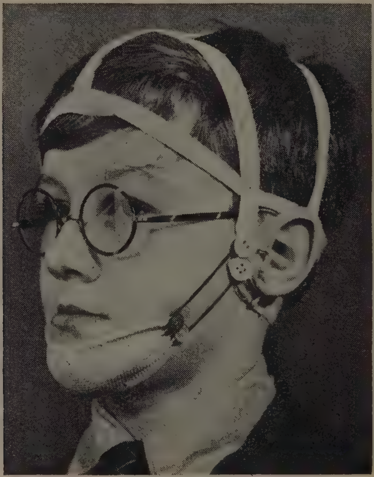

Fig. 372.—Patients wearing glasses suit their own convenience and comfort as to whether they wear them inside or outside the appliance. (E. S.)

If the protrusion is due to habitual thrusting forward of the mandible, the child must be broken of the habit, and a skull and chin cap, similar to that shown in fig. 372, must be worn day and night. Cases of this type are curable.

In cases due to faulty growth of bone, the treatment adopted must depend in a very great measure upon the cause and extent of the deformity.

Where the fault is traceable to the maxillæ and the deformity is slight, an expansion of the maxillary arch will often result in considerable improvement, and at times in complete correction of the deformity. Where the deformity is more marked it is desirable that, in addition to expanding the maxillary arch, an endeavour should be made to reduce the arch of the mandibular teeth by the extraction of an incisor or the first premolars. By this treatment it is often possible to make the arch of the maxillary teeth pass beyond that of the mandibular teeth, but, although some slight improvement in the expression may be gained, too frequently the treatment causes a complete disorganisation of the occlusion. Further, the maxillary teeth, even though they may be in a plane anterior to the mandibular, do not overlap, the result being that a retention appliance must be worn for a prolonged period to keep them in position.

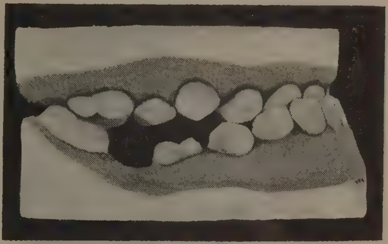

FIG. 373.

The case, the models of which are shown in fig. 373, will help to illustrate one or two points in treatment. The patient was a girl, aged fourteen. It will be seen that in complete occlusion there is a good deal of protrusion. In the mandible the permanent first molars have been removed and in the maxilla the left first premolar has been removed to make room for the permanent canine which erupted external to the arch. The maxillary and mandibular premolars fail to occlude, the result being that the whole process of mastication has to be carried on by the molar teeth. On examining the patient, it was noticed that the mouth was kept slightly open, the mandibular incisors being brought into contact with the maxillary. The patient stated that this was the most comfortable position, and that, during mastication, she constantly suffered from discomfort in the region of the articulation. An examination of the articulation with the teeth in occlusion, as shown in fig. 373, demonstrated that the condyle was brought forward, and was not resting in its normal position in the glenoid cavity. The protrusion was thus to a great extent artificial, and a fair result could be anticipated with proper treatment.

The lines on which treatment in such a case should be carried out are as follows: The anterior maxillary teeth should be brought forward and a biting apparatus inserted in the mandible.

In cases such as that shown in fig. 374 the deformity is greatly exaggerated by the loss of the posterior teeth. The insertion of artificial dentures will not only assist in restoring the masticating area, but, if the bite is raised, will hide the deformity to some extent.

When the protrusion of the mandible is so marked that the maxillary teeth bite completely within the mandibular arch, steps should be taken to procure a masticating area. This may be effected by the insertion of dentures, the exact plan to be followed in each case depending upon circumstances. In severe types of inferior protrusion, similar to those just referred to, it might be possible to improve the condition by surgical means. Several cases have been reported where surgical treatment has been adopted; one method being by bilateral symmetrical horizontal division of the ascending rami and the extra-oral wiring of the divided portions after the body of the mandible has been moved far enough back to bring the teeth into occlusion.

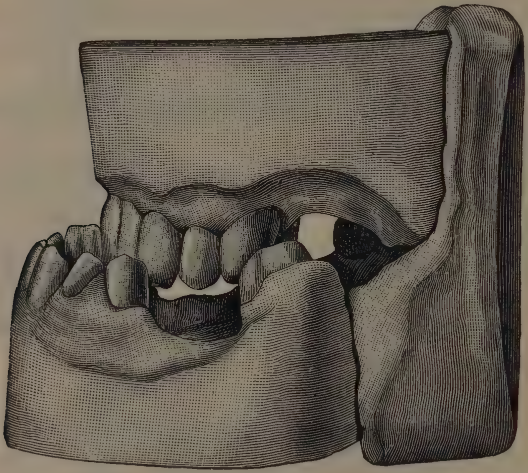

FIG. 374.

In considering the advisability of adopting resection of the mandible, the pros and cons must be carefully weighed. Resection of the mandible is always an operation attended by considerable risk, and even if the resected parts unite, the occlusion of the teeth is very likely to be so defective as to necessitate the use of dentures. It would therefore seem that by resection we cannot rely on gaining more than an improvement in appearance. Whether it is justifiable to submit the patient to the risks of a severe operation in the hope of gaining some improvement in personal appearance is a doubtful question.

When it is necessary to obtain a masticating surface, the better plan would certainly seem to be to disregard the question of appearance and treat the case by the insertion of dentures. In one case (a young girl) the question of resection was carefully considered, and the method suggested was the resection of the mandible in the median line in preference to resection on either side. Treatment on these lines would be attended with less risk than bilateral section of the mandible.

Cross Bite.—Cases in which there is unilateral protrusion of the mandibular teeth—a condition familiarly known as ‘ cross bite ’—may conveniently be included in this section. This deformity may be traced to habit or may arise from a defective growth of the maxilla, the ‘ cross bite ’ being acquired by the child in its efforts to obtain occlusion of the premolars and molars.

When the deformity is due to habit, an apparatus may be contrived to counteract the habit and so restore the normal occlusion. Where, however, the ‘ cross bite ’ arises from want of proper growth of the maxilla, an improvement may be obtained by an expansion of the maxillary arch.

Posterior lack of Occlusion.—Cases of this nature (see figs. 123 and 124) if seen during the eruption of the teeth may have the fault remedied by the use of an orthodontic appliance with spring force to move the misplaced tooth or teeth outwards or inwards into correct occlusion ; whether the maxillary or mandibular teeth are moved and whether they are moved buccally or lingually must depend on where the fault is deemed to lie. If seen too late in life to correct the lack of occlusion by orthodontic means it may be necessary, particularly if the condition is bilateral, to extract the non-occluding teeth in the jaw where the fault is deemed to lie and obtain occlusion and functioning of the opposing teeth by means of a denture.

(C) 'OPEN BITE.'

This term is applied to that abnormality in which some teeth only occlude when the mouth is closed.

From a clinical point of view 'open bite' may be considered under three groups :—

(1) Where the premolars and the molars occlude, but the incisal edges of the maxillary and mandibular anterior teeth are separated from one another by a space more or less oval in shape.

(2) Where the majority of the cheek teeth, as well as the anterior teeth, fail to occlude.

(3) Where there is lack of occlusion in the premolar and molar conditions. This condition may be unilateral or bilateral.

Groups (1) and (2) are often referred to as 'Anterior Open Bite' and group (3) as 'Posterior Open Bite.'

Etiology and Pathology.—In group (1) the deformity is often the result of an acquired habit, such as thumb-, finger-, or lip-sucking, the thumb or finger being bent and inserted horizontally between the incisal edges of the maxillary and mandibular teeth. The pressure thus exerted forces the maxillary teeth, as well as the alveolar process, in a direction upwards and slightly outwards. The mandibular teeth are forced downwards and backwards, but their displacement is usually less obvious than in the maxilla. In other cases there seems to be deficient bony growth in the premaxillary region, and this seems to be the cause when the condition occurs, as it commonly does in congenital syphilitics.

Cases included under group (2) vary greatly in the degree of their severity, so that while in some cases the deformity may consist merely of a slight separation between the teeth, in others it may extend to the condition shown in fig. 375. This type of 'open bite' is invariably accompanied by a marked narrowing in the region of the premolars and molars. In twenty-four measurements taken, the averages were 30·56 mm. between the first molars and 21·35 mm. between the first premolars. Compared with normal breast-fed children, the width across the molars and premolars is 100 : 88·4 and 100 : 80·7, respectively. The molar occlusion is not so abnormal as in superior protrusion, only a small percentage showing postnormal occlusion. Hypoplasic teeth are often present, the condition being sometimes very marked, and there is almost always a history of long-standing adenoids. The maxilla is defectively developed, the arch in the region of the molars and premolars being often within that of the mandibular teeth. In one section of these cases the opening increases progressively from behind forward ; in another section there seems to be marked bending down of the mandibular arch in the region of the mandibular permanent first molar ; while in a third section the mal-occlusion is considerably accentuated in front by an upward curvature of the maxillary incisors ; and a fourth section is found to combine the defects of sections 2 and 3.

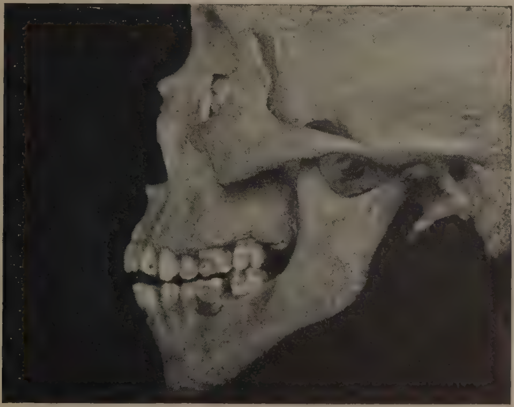

Fig. 375. (J. E. Grevers.)

Morbid anatomy specimens of ‘open bite’ are rare. Fig. 375 illustrates certain points noticeable in many of the cases, namely, an unusual growth of the anterior part of the mandible ; a short sloping ramus, and a marked bending of the mandible at a point near the spot where the facial artery crosses the bone and, in addition to, or in place of these, lack of development of the premaxilla or maxilla, or both, may be present. Anterior ‘open bite’ is a common but not a constant factor in children whose teeth are marked by the stigmata of congenital syphilis. The pathology of the condition is obscure. H. Schmidt, as quoted by H. Baldwin,[1] considers that when the mouth is kept open, as in marked nasal obstruction, the tongue is held down to the floor of the mouth and the tongue's tip is pressed against the anterior part of the mandible and teeth, exerting a downward and forward pressure. This pressure, operating in yielding bone, causes the alteration of the angle and so produces the deformity.

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[1] *Dental Record*, ix, 147.

The constant presence of adenoids and the association of the condition with hypoplasic teeth, which may be a sign of previous rickets, suggest that 'open bite' is due to the ill-effects of adenoids in conjunction with rickets. We know that the tension of the cheeks tends to narrow the jaw, and if this tension is acting on plastic bones the effect will be more pronounced. This may account for the marked narrowing of the arch. In severe cases the mandible takes a marked bend downwards, near the anterior border of the masseter, and this condition is probably due to a bending of the bones caused by the downward pull of the muscles which open the mandible, counteracted by the elevators of the mandible. The upward displacement of the maxillary incisors is possibly due to the prolonged action of the comforter, or the teat of the bottle, on bones softened by rickets. In other words, the agencies which produce general crowding and protrusion, when combined with rickets, produce grosser lesions which may show themselves in the form of 'open bite.' Deficient bony growth in the premaxillary region may also be produced in this manner so that the anterior teeth are unable to occlude.

The cause of *partial lack of occlusion* in the premolar and molar region is often obscure. In a few cases, faulty extraction may be responsible for the condition.

Treatment.

It is impossible to lay down any hard-and-fast rules for the treatment of 'open bite,' as each case must of necessity be considered on its merits.

Class (1), namely, **where the deformity is limited to the anterior teeth.** When seen at an early age, before the eruption of the permanent teeth, much may be done by breaking the patient of any vicious habits, and, with the eruption of the permanent teeth, the condition will be improved to some extent. When the habit of thumb-sucking, etc., has been continued during the eruption of the permanent dentition, or when the deformity is severe, treatment is of little avail. But it should be remembered that, although the appearance of the patient may be a little unsightly, the premolars and molars articulate, and the posterior teeth can, therefore, adequately perform their function of mastication. In these circumstances, it is generally better not to interfere, although, where the personal appearance is of the first importance, the crowning of the six anterior maxillary teeth might be deemed advisable. In a very severe case crowning would hardly be practicable, and it would be preferable to remove the anterior teeth and insert a denture. In cases due to deficient bone formation in the premaxillary region cutting in the bite is of value, particularly when the deformity is slight.

Class (2), namely, **where the majority of the posterior teeth fail to occlude.** A considerable improvement may be made in many cases by cutting in the bite. This method is extremely satisfactory, and may be employed alone or in combination with either extraction or the skull and chin cap. The treatment consists in stoning down the teeth until the premolars and molars occlude. This operation might with advantage be more generally adopted. The points in its favour are : (1) It gives the patient a good surface for mastication ; (2) it is permanent—teeth forced down by the skull and chin cap tend to rise again as soon as the apparatus is discarded ; (3) it relieves the patient of a long and tedious course of treatment.

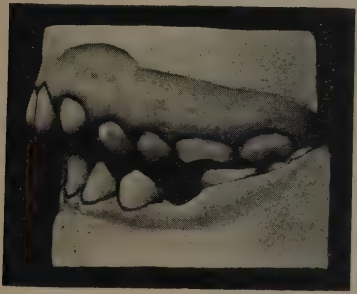

FIG. 376.

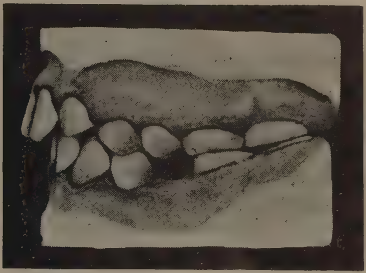

FIG. 377.

The operation should extend over several sittings, and a fair interval should be allowed to elapse between each. In the case shown in figs. 376 and 377 the treatment was spread over a period of one year. The object of leaving an interval between each sitting is that the pulp has time to react to injury caused by the operation, and the hypercalcification of the exposed dentine and the formation of secondary dentine which then take place allow a greater portion of tooth structure to be eventually removed.

The patient should be directed to apply spirits of wine to the cut surfaces twice a day, and the necessity of drying the surfaces thoroughly and keeping them entirely free from saliva for about a minute after the spirit has been applied should be emphasised.

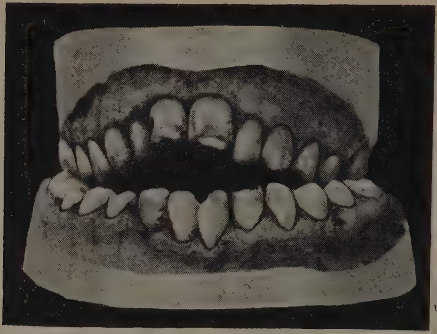

Fig. 378.

This treatment tends to diminish any sensitiveness which may exist. It is also a good plan to apply nitrate of silver to the cut surfaces for the same reason, but on account of its turning the dentine surface black it should not be used too near the front of the mouth.

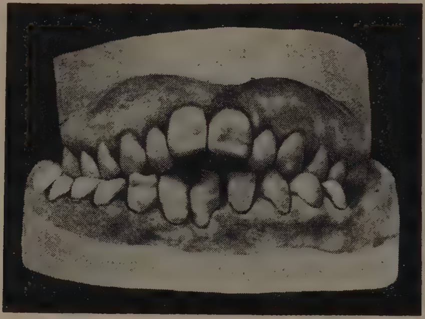

Fig. 379.

Another case treated by cutting in the bite is shown in figs. 378 and 379.

Where the deformity is only slight, an attempt may be made to correct the condition by the use of the skull and chin cap. If this treatment is to succeed, the apparatus must be worn constantly and the elastic bands stretching between the chin and skull caps arranged so as to exert pressure in an upward and not a backward direction.

In some cases the removal of teeth will be necessary. The extraction of the permanent second molars may suffice to remedy some examples, but if the permanent first molars are unsavable, the removal of the permanent second molars is hardly justifiable. Under such conditions, the following course of treatment will often yield satisfactory results. Remove the permanent first molars and use a skull and chin cap for a period of from six months to one year. If the occlusion is then still open, the operation of ‘cutting in the bite’ may be resorted to. Where the arch of the maxillary teeth is contracted, the occlusion may often be improved by tilting the molars and premolars outwards.

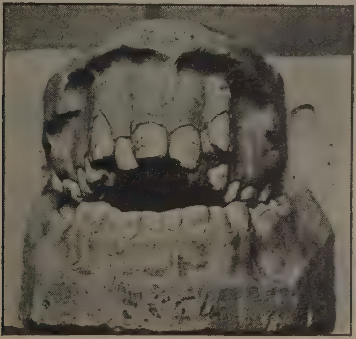

FIG. 380.—Patient, aged twenty-one, breast- and hand-fed fifteen months. Neave’s food. Tube feeding-bottle used. Adenoids present, not treated. Teeth hypoplasic. History of rickets; no history of use of comforter or thumb-sucking.

In severe forms extensive extraction must be resorted to, followed by the insertion of dentures. A typical case is shown in figs. 380 and 381. The teeth removed were $\begin{array}{r|r} 765 & 34567 \\ \hline 87\ 54 & 56 \end{array}$ and dentures were adapted with the result seen in figs. 382 and 383.

In the treatment of ‘open bite’ some practitioners adopt the plan of shortening the molars and lengthening the incisors, canines, and premolars; but such treatment cannot be relied on to give permanently good results. In connection with such treatment, it must be remembered that patients with ‘open bite’ often have an anterior gingivitis of long standing on account of the anterior teeth not being functional, and that the numerous bands and ligatures necessary for this treatment must inevitably increase the gum lesion ; indeed, it is probable that they expedite serious parodontal disease.

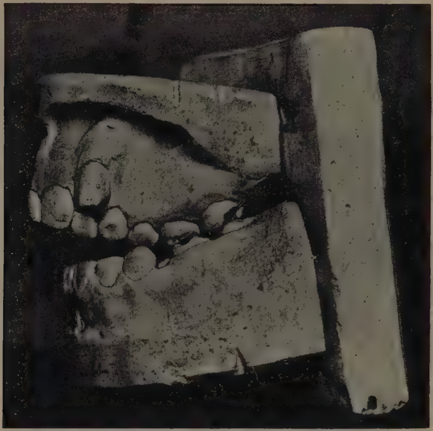

FIG. 381.

Cases of 'posterior open bite' may be dealt with by some form of mechanical appliance, but if occlusion cannot be attained by this means it may be better to extract the non-occluding teeth in the jaw judged to be mostly at fault and obtain occlusion and functioning of the opposing teeth by means of a denture.

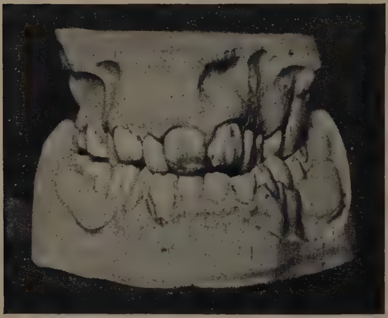

FIG. 382.

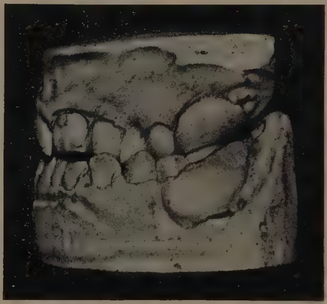

FIG. 383.

(D) ABNORMALITIES OF THE TEETH ASSOCIATED WITH CONGENITAL DEFECTS OF THE JAW.

This class of irregularities forms a small but well-defined group.

(i) Associated with Arrest in Development of the Mandible.—A partial arrest in the development of the mandible may occur unilaterally. The effect of the malformation on the dental arches will be to draw the teeth towards the affected side and cause the mandibular incisors to pass well behind the maxillary teeth, with the result that the lower lip will fail to cover the maxillary teeth and a marked protrusion of these teeth will follow. In treating such irregularities, ordinary orthodontic means are of little avail, and the most satisfactory means have been by means of removal of most or all of the mandibular teeth, the considerable deepening of the labial sulcus by means of an epithelial graft and the subsequent restoration of occlusion and chin by an appliance (denture) having a projection occupying the labial sulcus for the latter purpose. In this manner the extreme external disfigurement can be entirely done away with and a normal appearance produced.

(ii) Hare Lip.—Where the suture in the incisor region is involved, considerable irregularity of the incisors frequently follows, and R. A. Broderick [1] has shown that some cases of hare lip with no naked eye bony defect give radiographic evidence of a submucous bony cleft. Great caution should be exercised in attempting the correction of irregularities in such cases. The roots of the incisors are often malformed, and if treatment from an æsthetic point of view is necessary, the extraction of the teeth and the insertion of a denture is usually the wisest course to pursue.

(iii) Cleft Palate.—In cases of cleft palate involving the alveolar ridge the incisors adjacent to the fissure are often misplaced, and may erupt into the nasal cavity. As a rule, it is inexpedient to attempt their regulation, the roots of such teeth being usually curved and twisted. If the condition produces great disfigurement, the teeth can be removed and a denture inserted.

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[1] *Brit. Dent. Journ.*, May 15, 1935.

CHAPTER IX

Injuries of the Teeth arising from Trauma: Concussion, Dislocation, and Fracture.

It has been shown by one of us[1] that the trauma of mastication may produce minor damage either in the enamel only or in both the enamel and dentine. Such traumatic lesions do not *per se* give rise to any symptoms, and if not too extensive are repaired, though their results may almost invariably be seen on all teeth which have been used for any time if they are viewed in a good light, and in some people, particularly smokers, these traumatic fissures may become stained. The study of such lesions has shown that enamel and dentine are to some extent capable of repair, but such lesions are not at present of practical concern to the dental surgeon.

Those traumatic lesions which concern the practitioner are as follows :—

(A) CONCUSSION.

Concussion may give rise to slight or severe periodontitis, and may be complicated by inflammation of the pulp, which may lead to death of the pulp. The pulp may be ruptured from its connections at the apical foramen, and when this occurs the pulp often remains quiescent, but marked discoloration of the tooth substance usually supervenes, much more rapidly in children, where the dentinal tubes are more patent than in adults.

(1) Causes.—Concussion of the teeth may arise from direct or indirect violence. A blow on the mouth from a fist or cricket ball or biting unexpectedly on any hard substance are examples of direct violence, and a blow on the chin, a fall or jumping upon the heels instead of upon the toes are examples of indirect violence.

(2) Signs and Symptoms.—In most cases of concussion of the teeth the patient will be found to be suffering from periodontitis more or less severe. Where the pulp has not been ruptured at the apical foramen, or the inflammation of the periodontal membrane has not extended to the pulp, the tooth will be found to be sensitive to pressure and slightly loose, and will respond to thermal changes in the same way as the normal tooth. Should the inflammation spread to the pulp, the tooth will become exquisitely sensitive both to pressure and to slight variations of temperature, the periodontal pain being dull, constant, and restricted to the tooth, while the pain arising from the inflamed pulp will be of a sharp lancinating character, and will be referred at times to other teeth and adjacent parts. When suppuration in the pulp occurs, the tooth will no longer be sensitive to changes of temperature, but the periodontal inflammation will increase in severity owing to the suppuration having spread to the periosteum. The pain will be of a throbbing character.

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[1] *Proc. Roy. Soc. B.*, 1930, 106, 376. (E. S.)

(3) Treatment.—The treatment will depend upon whether or not the pulp is involved in the injury. *When the periodontal membrane only is affected*, local depletion will be found efficacious ; in mild cases, counter-irritation, with capsicum plasters, tinct. iod., will be sufficient. *When suppuration of the pulp has taken place*, the pulp chamber must be opened in the direction which will render removal of the pulp most easy ; the dead pulp must be removed and the canals treated. Where concussion has caused pulp irritation, and at the end of two or three days no improvement is apparent, the pulp should be removed, as prolonged efforts to save the pulp are nearly always followed by periodontitis of an intractable type. The following case is instructive : A patient, aged twelve, fell and fractured the maxillary right first incisor, and lacerated the pulp. The left first incisor was implicated by concussion. The pulp from the right first incisor was removed, and attempts made to save the pulp in the left tooth, which was irritable. The periodontitis around the right tooth rapidly cleared up, but continued with the left tooth. As the pulp condition failed to disappear at the end of three weeks, the canal was cleared of the morbid pulp, sterilised and filled. Chronic periodontitis continued, in spite of repeated and various treatments, for four years, so that removal of both teeth was decided upon. A radiogram of the teeth showed that the root of the tooth originally fractured had been attacked by rarefying periodontitis on the aspect adjacent to the left tooth, and the root of the latter had, to a great extent, disappeared from a similar periodontal condition. If the pulps of the teeth had been removed within a few days of the injury it is probable that the periodontitis would have passed away and the teeth would have been permanently saved.

(B) DISLOCATION.

(1) Erupted Teeth.—Dislocation is displacement of a tooth from its normal position in its socket. Dislocation may be incomplete or complete—that is, a tooth may be forced into, loosened in, or totally displaced from its socket.

(*a*) **Causes.**—Dislocation, like concussion, is the result of violence, but the injury causing it is usually of a more severe nature.

(*b*) **Treatment.**—*When the dislocation is incomplete*, the tooth should be replaced in its normal position, the broken alveolar process moulded around it, and counter-irritants applied to the gum. With all dislocations, precautions should be taken to keep the tooth fixed, either by ligatures of silk or wire, or better, by a splint made of a suitable metal. *If the tooth is driven into the socket*, it should be grasped with a fine-bladed pair of forceps and carefully drawn into place. When a tooth is driven upwards towards the maxillary sinus, it is probably a safer plan to leave it alone rather than attempt its removal. If removal is considered advisable, care must be taken to prevent the complete displacement of the tooth into the maxillary sinus in the attempt at removal.

If the displacement is complete, the treatment will vary according to the age of the patient and the time at which the case is seen subsequent to the accident. In the young, if seen immediately after the accident (as, for example, when the tooth is displaced in the operating room), the tooth should be replaced at once, as under these conditions union of the structures entering at the apex may take place (see pp. 415 and 889). If the patient is an adult, or is seen some hours after the accident, the pulp of the injured tooth must be removed and the canals sterilised and filled before the tooth is replaced.

(2) Unerupted and Incompleted Teeth.—Teeth may be injured in their crypts by trauma or after eruption before the root is completed, the portion of the tooth already calcified being displaced from the portion which is developing. It occurs very much more commonly in the permanent dentition. If the injury does not destroy the vitality of the tooth calcification progresses and a condition known as *dilaceration* is ultimately produced. (See also Traumatic Complex Odontomes, p. 740 and p. 42.)

The calcified portion of the tooth is usually dislocated in such a manner as to form an angle with the developing portion (see figs. 384 to 388). In the case of premolars the calcified

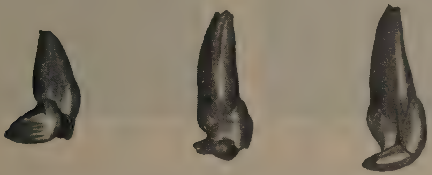
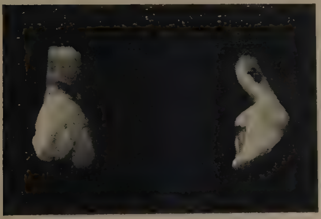


FIG. 384.—Maxillary first incisors, showing the results of injury during development of the crowns.



(*a*) (*b*)

FIG. 385.—Maxillary permanent (*a*) first and (*b*) second incisors, showing the results of injury during development of the roots. (London Hospital Dental Museum.) (Sir Francis Farmer and E. S.)

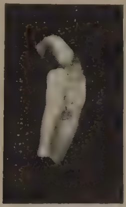
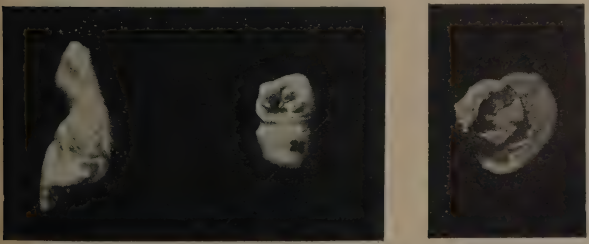


FIG. 386.—Mandibular permanent incisor, showing the results of injury during the development of the crown. (London Hospital Dental Museum.) (J. Noriskin.)



(*a*) (*b*) (*c*)

FIG. 387.—(*a*) Maxillary permanent canine, (*b*) mandibular second premolar, (*c*) mandibular third molar, showing the results of injury during the development of the roots. (*c*) was caused by instruments in removing an impacted mandibular permanent second molar at age 10-6; the specimen was removed six years subsequently. (London Hospital Dental Museum.) (Sir Francis Farmer and E. S.)

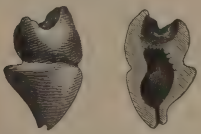


FIG. 388.—Mandibular premolar. (G. G. Campion.)

portion occasionally appears to be forced into the developing part, causing a kind of impacted dislocation. It is possible that this condition is at times due to careless removal of the deciduous teeth or to inflammation and/or pressure having origin from diseased deciduous predecessors (see p. 42 and fig. 50). When the causative trauma is of this gentler and more constant nature the deformity produced may be of the nature of a bend or twist in the direction of the root, and bends and twists may also be brought about by a lack of room owing to crowding (fig. 389). The injury to the growing tooth may result in an arrest of growth. Dislocation of unerupted teeth is more common in the anterior teeth, since they are more exposed to injury. Traumatic injury to the developing permanent canines is rare owing to the position they occupy in the bone, and dilaceration of these teeth is very rare on account of their state of calcification on eruption (fig. 387(*a*)).

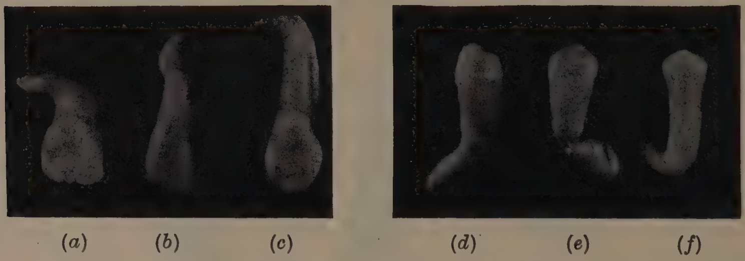


FIG. 389. (From Bennett's 'The Science and Practice of Dental Surgery,' *Oxford Medical Publications.*)

(*a*) Maxillary permanent first incisor with markedly curved root.
(*b*) Maxillary permanent second incisor with two curves in root.
(*c*) Maxillary permanent canine with markedly curved root.
(*d*), (*e*), (*f*) Mandibular premolars with marked curves in roots.
(London Hospital Dental Museum) (E. S.)

FIG. 390.

The injury may occur either to the crown or to the root, according to the state of development of the tooth at the time of injury.

A case was recorded by J. Lewin Payne[1] in which the mandibular right first premolar was in two pieces, as shown in fig. 390. The upper part of the lower fragment was cone-shaped and fitted accurately into the hollowed-out base of the upper fragment. The pulp cavity in the upper fragment was completely obliterated by 'adventitious dentine,' the pulp in the lower fragment being uncalcified. It seems possible that the condition of the tooth was the result of trauma.

Occasionally the pulps of newly erupted teeth which have been loosened by trauma will continue functional till calcification of the tooth is complete, and then die.

A ground section of a dilacerated tooth will show a sudden bend in the dentine tubes at the junction of the tissues formed before and after the injury; interglobular spaces may also be found in the dentine first formed after the injury, and the cementum may be thickened at the site of injury.

(C) FRACTURE.

Fractures of the teeth vary from slight cracks or fissures of the enamel to extreme comminution of the whole tooth tissue.

They may be grouped as follows :—

- (*α*) Fractures of the crown.
  - (i) Without loss of tissue.
  - (ii) With loss of tissue.
    - (*a*) Not involving the pulp.
    - (*b*) Involving the pulp.
- (*β*) Fractures of the root.
- (*γ*) Fractures of both the crown and the root.
- (*δ*) Comminuted fractures.

(1) Causes.—The causes of fractures are direct or indirect violence.

With advance in age increased calcification of the tissues of the teeth takes place, and this renders the teeth more brittle, and therefore more liable to fracture. This accounts for the fracture of teeth which occasionally occurs in elderly people during mastication. An example may be quoted. A man, sixty years of age, during mastication, fractured the maxillary left first premolar. He possessed the full complement of teeth, and there was no caries and very slight parodontal disease. The tooth was split in a mesio-distal direction through the fissure on the occlusal surface, the split continuing in a vertical direction upwards to the bifurcation of the roots. An examination of the tooth after extraction showed extreme translucency of the tissues, indicating considerable calcification of the originally soft tissue of the dentine.

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), v, 141.

Cases of fracture from the force of mastication are not always readily detected owing to the fragments being held tightly together by the surrounding structures. Under such conditions the pulp may respond in a normal manner to thermal changes and to percussion, the only symptom being an acute pain at times during mastication.

(2) Treatment.

Group (*a*)—*Without loss of tissue.*—In these cases the enamel, and possibly the underlying dentine, is slightly cracked or fissured ; as there is no loss of substance no treatment is required unless there is accompanying periodontitis.

With loss of tissue and not involving the pulp.—If the fracture involves only enamel, it will be necessary to smooth the rough edges with suitable instruments and polish the surface. Should the dentine be exposed, it may be found hyper-sensitive, especially in young patients. There may also be hyperæmia of the pulp, characterised by extreme sensitiveness to heat and cold, and pain on pressure owing to the congestion extending to the periodontal membrane around the apex. The treatment is to employ counter-irritants or local depletion, at the same time to apply to the exposed dentine local remedies, such as absolute alcohol, nitrate of silver, or chloride of zinc, or even to protect the exposed dentine by cementing on a metal cap. The symptoms usually subside, and at a later period the tooth and its neighbour should be trimmed in such a way as to reduce the disfigurement as far as possible or, should restoration be desirable for æsthetic reasons, as in anterior teeth, it is best done by means of porcelain inlays or jacket crowns. Should suppuration occur in the pulp from continued irritation, the pulp cavity must be opened and treated in the usual way. In incompletely calcified teeth every effort must be made to retain the vitality of the pulp.

With loss of tissue and involving the pulp: Maxillary first incisor.—The treatment will depend upon—

(i) The stage of the development of the root.

(ii) The sex of the patient.

(iii) The presence or absence of a crowded condition of the teeth.

(i) *The Stage of the Development of the Root.*—A radiogram should always be obtained if there is any doubt as to the stage of formation of the root, as the time when the apex of any tooth is completed varies considerably within the range of normal.

If the formation of the root is incomplete the tooth should be removed. The question then to be decided is whether it is better to allow the space to close and to disregard the personal appearance, or to retain the space for purely æsthetic reasons, a denture or bridge, with its attendant disadvantages, being inserted at a later period. When fractures occur after the completion of the root, the pulp should be removed and the canals treated; crowning can then be carried out at a future date.

(ii) *The Sex of the Patient.*—The loss of an incisor to a boy is of less æsthetic importance than to a girl.

(iii) *The Presence or Absence of Crowding of the Teeth.*—Where there is doubt whether to save or remove a fractured tooth, the condition of the teeth as regards crowding is an important consideration. If the teeth are crowded there is a reasonable probability that the space created by the loss of the tooth will close up completely, but if there is no crowding it is highly probable that the space will remain permanently. Each case must be treated on its merits. The following comments may be useful.

In *fracture of a maxillary permanent first incisor in a boy before the root is fully formed*, removal of the tooth is to be recommended. In a boy the æsthetic question does not outweigh the advantages of avoiding a denture.

Fracture of a maxillary permanent first incisor in a girl before the root is fully formed.—The tooth should be removed and the interval retained by an appliance attached to the approximal teeth (fig. 391). In constructing this appliance, care must be taken that the bands around the teeth are clear of the gingival margin. A denture or bridge should be inserted when the premolars and permanent canines are in position.

In making dentures for this type of case, the best method is to attach the artificial tooth to a thin vulcanite plate which should cover the greater part of the roof of the mouth, thus avoiding all kinds of clasps.

The disadvantage of a small metal plate is that it is necessary to retain it by means of clasps, and as the clasps must be taken between the teeth the interproximal spaces are damaged.

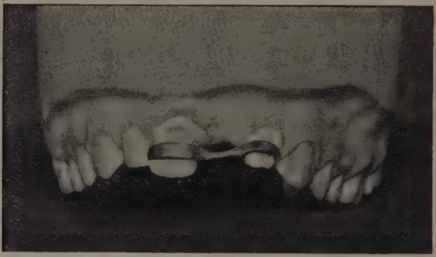

FIG. 391.

Fracture of a maxillary permanent first incisor after the completion of the root.—If the teeth are crowded, the better plan is to remove the fractured tooth. In the case of a boy the approximal teeth should be allowed to close together, but in the case of a girl the space should be retained. If the fracture is in the position shown in the diagram (fig. 392) the neighbouring teeth will not tend to approximate. If the fracture is near the region indicated by the dotted line, there will be a tendency for the neighbouring teeth to approximate. Steps must be taken to keep them apart by means of a stay fixed in the pulp chamber (fig. 393).

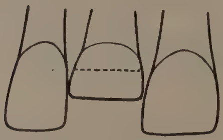

FIG. 392.

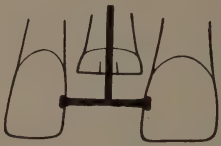

FIG. 393.

Maxillary Permanent Second Incisor.—Where the tooth is fractured before the root is fully formed extraction is advisable. In the case of a girl the appearance of the slightest crowding of the teeth on the opposite side of the mouth should be relieved in order to prevent as far as possible the centre of the mouth shifting (see p. 150). Fracture subsequent to the period at which the growth of the root is completed should be treated by removal of the pulp and the filling of the pulp canal. The tooth can be crowned at a suitable age.

In cases of fractured maxillary second incisors in crowded mouths, the removal of the tooth may be advisable. Judgment must be guided by the sex of the patient and the direction of the root of the permanent canine.

Mandibular Permanent Incisor.—In almost every case removal is best. The space, even if it does not completely close, is not very noticeable.

Fracture of a permanent canine, premolar, or molar should be treated by removal of the pulp and restoration of the tooth by suitable means.

Groups (β) *and* (γ).—Fractures of the root, or the crown and root, should be treated by extraction.

Group (δ).—Cases of comminuted fracture are generally caused by severe violence. Several teeth are usually involved, and the alveolar process is generally fractured. In these cases it is better to reflect the muco-periosteum from the surface of the bone and obtain a view of the area of fracture. The fragments of teeth and bone can then be removed and the muco-periosteum returned and secured in place by sutures.

(3) Healing of Fractured Teeth.—Union of fractured teeth may take place, and a few cases have been recorded. The union is similar in character to that which takes place in bone. In a case quoted by Wedl, and shown in his 'Atlas of Pathology,' [1] union was effected by means of the periodontal membrane and the pulp.

A rare example of union of a fractured tooth was recorded by Storer Bennett.[2] A maxillary incisor had been fractured across the crown. The patient was seventeen years of age, and ten months previously had fallen down, struck the tooth and dislocated it upwards, when it became impacted and remained until removed owing to pain. The tooth is shown in fig. 394. The bond of union is of a cavernous character with numerous spaces for blood-vessels (see fig. 395).

Fractured roots are more likely to unite when the fracture is near the apex, because the fragments are kept in fairly accurate apposition by the socket of the tooth. An excellent example is recorded by G. W. Watson.[1] The maxillary right first incisor of a man, aged twenty-four, was loosened by a blow from a cricket ball. The tooth remained loose and painful for a month after the injury and was slightly misplaced inwards, but eventually became firm and serviceable again. About six years later the tooth was removed, and it was then seen that the blow had fractured the root and repair had followed. The appearance of

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[1] 'Atlas to the Pathology of the Teeth' (Second Edition), 1893, figs. 107 to 109.

[2] *Trans. Odonto. Soc.*, xxviii, 181.

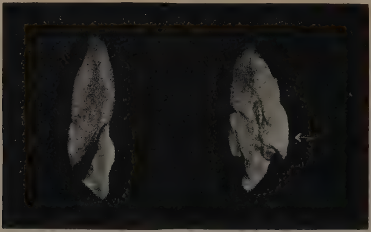

FIG. 394.—The tooth from which the section (fig. 395) was made.

(From *Trans. Odonto. Soc.*)

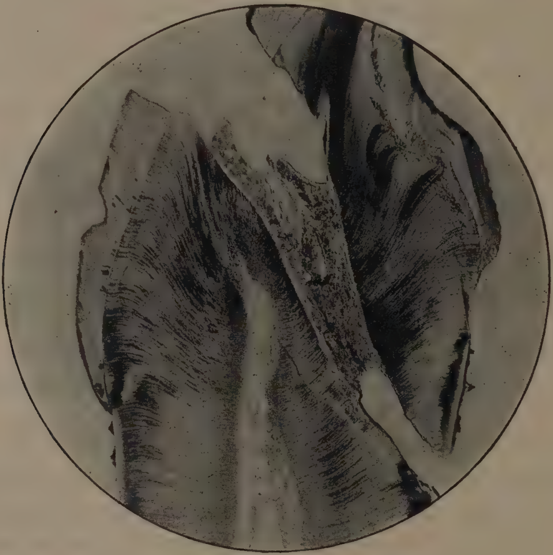

FIG. 395. (F. J. Bennett.)

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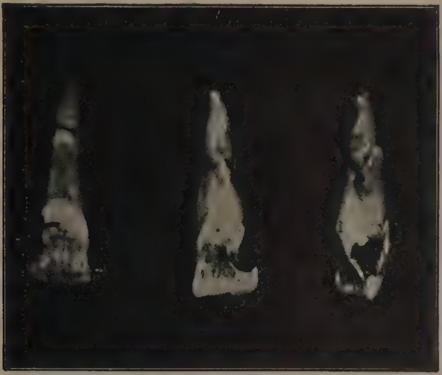
[1] *Dental Record*, May 1906, 162.

the tooth was as follows : ‘About a quarter of an inch from the apex of the root on its labial surface there was a deep, somewhat irregular crescentic groove, continued half-way round the median



Anterior Posterior Distal
surfaces.

Fig. 396. (From *Dental Record.*)

and distal surfaces. Radiating from these two points and extending round the lingual surface of the tooth was a series of comminuted fractures running in all directions.’ Illustrations of the tooth are shown in fig. 396.

CHAPTER X

Caries of the Teeth.

Prevalence of Caries—Frequency in Individual Teeth—Morbid Anatomy and Pathology—Experimental Reproduction of Caries—Susceptibility and Immunity to Caries—Etiology—Symptoms—Spontaneous Arrest of Caries.

The teeth of man have been affected by caries from the earliest times. The Rhodesian skull, belonging to a race which existed long before the present type of human beings was evolved, shows caries of many teeth.[1] It also shows much attrition and some alveolar absorption.

(A) PREVALENCE OF CARIES.

(1) Ancient Races.

J. R. Mummery [2] examined skulls from 'the ancient British tumuli of Wiltshire.' In sixty-eight skulls (dolichocephalic type) from long barrows, which date back to the *Stone Age*, only two cases of caries were discovered, one on an approximal surface and one on an occluding surface. These people were a pastoral and not an agricultural race. They lived by the chase and their customs were barbarous.

In thirty-two skulls (brachycephalic type) from round barrows (*Bronze Period*), there were seven cases of caries, six on approximal, and one on occluding surfaces. These people probably consumed mostly vegetables in the form of coarsely prepared barley or oats. (This race was referred to by Cæsar in his 'Commentaries.')

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[1] Smith Woodward, *Proc. of Anat. Soc. of G. B. and I.*, May 1921–Feb. 1925, 22; and *Brit. Dent. Journ.*, 1921, 1156.

[2] *Trans. Odonto. Soc.*, ii, N.S. Over 3,000 skulls were examined and, of these, 1,658 were tabulated, the remainder being rejected either in consequence of their doubtful authenticity, or because too many teeth were missing. This work of J. R. Mummery entailed a vast amount of labour, and is one of the most valuable contributions to the dental literature of the 'sixties.

In sixty skulls from Yorkshire barrows, consisting principally of the earlier or long-headed race, twenty-four exhibited caries, and in nine cases the disease was extensive. In some cases the incisors were affected. Nearly half these skulls showed extensive attrition of the teeth, and there were thirteen examples of dento-alveolar abscess.

The skulls of *Romans* found in England also furnish ample evidence of caries. In 143 skulls caries was present in forty-one cases; in nineteen skulls more than six teeth were affected and in twenty-nine skulls caries was present on approximal surfaces. In some cases all the teeth were affected, including the incisors. The diet included meat, fish, wheat and miscellaneous luxuries. It is of interest to note that twenty-three of the skulls were found in Yorkshire, and of these eighteen exhibited caries.

Passing to the *Anglo-Saxon* period, we find that of seventy-six skulls, caries was present in twelve; in five instances the approximal surfaces were attacked.

Egyptian mummies to the number of thirty-six were examined; fifteen showed caries—in nine cases all the premolars and molars were affected, and in two cases all the maxillary incisors.

J. G. Turner examined 104 Egyptian skulls belonging to the period of the twenty-fifth to the thirtieth dynasties: 40·38 per cent. showed caries, and, of 435 loose teeth, 16·6 per cent. were affected.

The foregoing statistics show that races like the Romans and Egyptians which had reached a high standard of civilisation, had a greater incidence of caries than races living under more primitive conditions.

(2) Existing Primitive Races.

In an examination of existing primitive races, Mummery found evidence of caries in all of those of which statistics are given.

The following is a résumé of the section of Mummery's paper dealing with existing primitive races [1]:—

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[1] The term 'extensive' is used for skulls showing over six carious teeth.

Eskimo.—A nomadic people inhabiting the littoral in the extreme north. Diet: Almost entirely meat, fowl, and fish, of which huge quantities—10 to 14 lb. per person—are consumed daily. Only one case of caries in sixty-nine skulls. In another instance two molars were missing.

Indians of North-West Coast of America.—Diet: Chiefly dried fish. Two cases of caries in fifty-one skulls. The approximal surfaces of two teeth were attacked. The fish is coarsely prepared for food and quantities of sand adhere to it. The teeth show considerable attrition.

North-American Indians (*interior*).—Often subject to great privations. Diet: Meat with occasional addition of roots. Two cases of carious teeth in twenty-one skulls.

Gauchos.—Mixed race of Indian and Spanish blood in the Argentine Republic, occupied on ranches and living mostly on horseback. Diet: Entirely 'roast beef'; sole beverage, Paraguay tea without sugar. No record as to caries is given, and only one case of toothache was discovered.

Indians of the same race, inhabiting towns and indulging very freely in artificial diet, such as acid confectionery and inferior wines. Caries prevalent.

Fiji Islanders.—Addicted to cannibalism. Diet: Meat, fish, maize, bread-fruit, plantain, coco-nut. Two cases of caries in thirty-eight skulls; in one, two teeth were affected and, in the other, caries was extensive.

Maori.—Addicted to cannibalism. Diet: Meat, fish, fruit, and vegetables. According to Pickerill, their staple food was the kumara or sweet potato. Two cases of caries in sixty-six skulls; in one of which two teeth and, in the other, four teeth were affected on the approximal surfaces.

In 260 skulls of the uncivilised period, examined by Pickerill, two cases of caries; but out of fifty children living under European conditions 95 per cent. showed caries.

Inhabitants of Eastern Polynesia.—Diet: Pork, fish, coco-nut, plantain, bread-fruit, and sugar-cane. Eight cases of caries in seventy-nine skulls, most of them extensive, and in one case the approximal surfaces were affected.

Sandwich Islanders.—Diet: Mainly vegetable with a little meat; staple food a root called 'taro,' roasted and eaten as a substitute for bread, to which sweet potato and sugar are occasionally added. Four cases of caries in twenty-one skulls, two being extensive; in three cases the approximal surfaces were affected.

Australian Races.—'For the most part improvident savages, and spending their lives in alternate feasting and famine.' Diet: Mainly meat, fowl, fish, and in times of scarcity lizards, ants, frogs, and wild roots. Caries in twenty-seven skulls out of 132, the approximal surfaces being affected in fifteen cases.

Tasmanian Race.—Closely resembling the Australian races in habits and diet. Caries in nine skulls out of thirty-three, mostly affecting a considerable number of teeth, and in eight cases affecting the approximal surfaces.

Zulu Kaffirs.—Staple food: Coarse-ground mealies, meat being consumed on special occasions, as in the training of their warriors. Caries in six skulls out of forty-nine; five of very limited extent, one case of approximal caries.

African Tribes supplying the Slave Markets.—The most feeble of the African tribes; mainly in unhealthy districts. Diet: Mixed, chiefly vegetables. The skulls of 268 slaves who had died of disease or exhaustion were examined. In sixty-six caries was present, many of the cavities being on approximal surfaces. In no less than sixteen of the cases the whole of the molars and premolars were carious.

The Bushmen.—Dwarfs inhabiting the deserts of South Africa. Diet: Chiefly meat, but in times of scarcity edible roots, locusts, etc. In twenty-nine skulls six cases of caries were found, the majority being extensive. In three cases the approximal surfaces were affected.

The Natives of Southern India.—Diet of a varied character, frequently of an unwholesome nature. Rice forms the staple food; sweetmeats are much indulged in. In seventy-one skulls ten cases of caries, in three of which six teeth were involved. Four cases of approximal caries.

The Natives of Northern India.—Diet: Vegetables of a simple and wholesome character, staple food being wheat. In 152 skulls nine cases of caries, and in no instance more than two teeth affected. One case of approximal caries.

Malays.—Diet: Mixed; fruit, birds, and fish. In twenty-four skulls three were affected, and in one of these there was extensive caries.

Chinese.—Staple food, rice. In ten skulls out of twenty-seven caries was present, and in six the disease was extensive.

Mummery's results in tabulated form are as follows :—

	Skulls examined.	Percentage of skulls showing carious teeth.	Skulls showing destructive attrition.
Eskimo	69	1·4	22
Indians N.W. Coast North America	56	3·9	32
Indians interior N. America	23	9·5	2
Fiji	38	5·2	4
Maoris (New Zealand) ...	67	3·0	25
Eastern Polynesia	79	11·4	15
Sandwich Islanders ...	21	19·0	4
Australian Races	132	20·4	28
Tasmanian Race	33	27·2	6
Kaffirs (Zulu)	49	14·2	6
African Slaves	236	20·8	23
Bushmen	29	20·6	9
Southern Indian Races ...	71	14·0	10
Northern Indian Races ...	152	9·5	8
Malay Races	24	12·2	2
Chinese	27	37·0	0

Mummery's figures show that races whose principal diet is flesh are more free from caries than those existing wholly or mainly on a vegetable diet. Exception has been taken by certain writers to the dietaries set forth by Mummery. In this connection it is to be noted that the present-day diets of primitive races are not necessarily the same as those of some years ago. For example, the Barotze (N.W. Rhodesia) within the last seventy years have added cassava (a root from which our tapioca is made) to their staple food-stuffs.

S. Colyer has published statistics relating to certain *African tribes of Bantu descent* :—

Central African tribes from the region of Lakes Mweru and Bangweulu.[1]—The figures were provided by 1,000 natives (males) examined consecutively. The natives came from three districts, which will be referred to as I, II, III. Districts I and II were 'saltless,' District III 'a great salt producer.' The natives were divided into three classes according to age :—

Decade 2, ten to twenty years.
,, 3, twenty to thirty years.
,, 4½, thirty to forty-five years.

The division must be regarded as approximate, the ages having to be guessed.

Many of the natives file the front teeth and expose the dentine, which in many cases shows caries. These teeth are not included in the following figures, because the object of the examination was to discover the incidence of true caries of the enamel. The following table shows the percentage of caries in the different age periods :—

---

[1] *Dental Record*, xxxvii, 407.

	Decades.			
	2	3	4½	Total.
Number of individuals examined … …	246	670	84	1000
Percentage with caries … … …	5·7	9·8	21·4	9·8
Percentage without caries … … …	94·3	90·2	78·6	90·2
Percentage of carious teeth per individual	·3	·7	2·1	·73
Percentage of teeth per individual lost from other causes … … … …	·1	·3	3·7	·58

Note.—All teeth the loss of which was probably due to caries are included as carious teeth. The number of teeth per individual is taken as twenty-eight.

The incidence according to districts is shown in the following table :—

District.	Number of individuals.	Percentage with caries.	Percentage of carious teeth.	Percentage of teeth lost from other causes.
I	185	8·1	·36	·42
II	700	11·1	·91	·67
III	115	5·2	·24	·21

Note.—District III is the salt-producing area for the surrounding country.

The diet of these natives is mainly millet porridge, to which is added in the rainy season maize, pumpkins, beans, and potatoes.

South-East African tribes in the Transkeian territories.[1]—These natives, of Bantu descent, have caries similar to that of highly civilised man, that is, interstitial and fissure cavities. Natives may be seen with ‘every tooth save, perhaps, a few isolated ones in their jaws, decayed and level with their gums.’ Caries of the incisors is comparatively common.

The staple foods are mealies and Kaffir corn ; sugar is consumed in large quantities, taken with porridge, used to flavour tea and coffee, and eaten raw. One family of six members consumed six pounds of sugar each week.

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[1] *Dental Record*, xxxix, 401.

In investigations on caries, it is important that the position of the cavities should be given and a distinction made between caries starting in the enamel and in the cement.

The following figures given by J. G. Turner relative to *South Sea Islanders* show the high degree of cervical caries in some of the primitive races. Their diet is fruit, vegetables, and coconut coarsely prepared. Turner found caries in 28·88 per cent. of ninety skulls and 8·24 per cent. of 1,116 teeth. There were thirty-five interstitial-cervical, twelve lingual-cervical, and twenty-four buccal-cervical cavities. Ninety teeth had been lost ante-mortem.

An interesting report on the examination of old human crania deposited in the principal museums of the United States is published in the *Transactions of the American Dental Association*, 1894, and is well worthy of perusal by those wishing to investigate the subject. The following is a résumé of the results obtained :—

Total number of teeth examined	46,657
Total number of teeth diseased	11,338
Percentage of diseased teeth	24·3

The crania were grouped geographically as follows :—

	Number of teeth examined.	Number of carious teeth.	Percentage of carious teeth.
South America	6,719	930	5·8
Central America	930	44	4·87
North America	27,362	1,394	5·09
Europe (including Anglo-Americans)	3,422	242	7·07
Pacific & Sandwich Islands	2,738	118	4·3
Egypt (including Africans)	3,306	113	3·41
Asia	2,180	45	·06

(3) Modern Civilised Races.

Among modern civilised races the percentage of mouths containing carious teeth has increased very considerably.

Londoners of the Seventeenth and Eighteenth Centuries.—The following facts obtained from an examination of a series of skulls and mandibles belonging to the seventeenth and eighteenth centuries afford some evidence of the condition of the teeth of Londoners of that period :—

	Specimens examined.	Number with full dentures.
(1) Skulls with mandibles	38	10
(2) Skulls only	128	48
(3) Mandibles only	227	103

In the specimens 3,349 teeth were present, 345 being carious, or 9·8 per cent. Dividing the specimens into two groups—

(*a*) those in which no teeth had been lost from disease,
(*b*) those showing loss of teeth from disease,

we find that in group (*a*) 1,449 teeth were present, of which fifty-nine were carious, or just over 4·07 per cent.; in group (*b*), 1,900 with 286 teeth carious, or about 15 per cent.

The post-mortem loss of so many teeth, especially incisors, canines, and premolars, in old skulls makes it very difficult to arrive at a reliable estimate of the amount of caries, so that data obtained from the molars, and especially the permanent first molars, give a truer picture of the amount of caries in these teeth. Of 1,456 first, second, and third molars, 212 were carious, or 14·5 per cent. Of 581 permanent first molars, 105 were carious, or 18 per cent.

In order to obtain a more accurate estimate of the probable number of teeth which had been attacked by caries, it is necessary to—

(1) Include as carious teeth a proportion of those lost ante-mortem. Half the teeth were considered to have been lost from caries, the other half from parodontal disease.

(2) Estimate the number of carious teeth among those lost post-mortem.

Working on these lines, it was estimated that of the 6,804 teeth, approximately 14·6 per cent., and of 862 permanent first molars, 27·6 per cent., had been attacked by caries. An examination of the series of thirty-eight skulls with mandibles showed:—

Number of skulls and mandibles.	Number of carious teeth.
12	0
11	1
4	2
2	3
2	5
3	6
1	9
1	11
1	13
1	17

In other words, fifty of the 103 carious teeth were present in four specimens.

The collective investigations of the British Dental Association showed that in the mouths of 10,500 English and Scotch girls and boys, averaging twelve years of age, there were no less than

37,000 unsound teeth (deciduous and permanent). In all, 86 per cent. of the mouths showed that caries was present in one form or another.

The subjoined table[1] shows the relative ratio per hundred of children with unsound teeth, defective deciduous teeth, and defective permanent teeth. The figures afford an illustration of the early age at which caries begins, and also of the progressive increase of the disease as children grow older.

Age period	iv.-vi.	vii.-ix.	x.-xii.	xiii.-xv.	xvi.-xviii.
Number examined ..	744	1,716	3,071	2,367	268
Sound (no decay)	23·8	14·2	16·1	14·1	6·4
Defective deciduous teeth only	67·4	43·3	18·3	5·1	0·1
Defective permanent teeth—					
1–4	8·8	41·5	55·9	51·9	37·3
5–8	—	1·9	8·5	22·9	32·6
9 or more	—	—	1·2	6·0	23·6

Another illustration of the ravages of caries can be gathered from the statistics compiled from the examination of the children in the county of Shropshire.[2] It was found :—

(i) That only 3 per cent. of the children at the age of twelve and only 5 per cent. at the age of five were free from caries.

(ii) That the average number of carious teeth at the age of five was 6·8, and at the age of twelve was 4·8.

(iii) That of 3,794 children examined at the age of five, no less than 1,017 had ten or more carious teeth ; and of 3,580 children at the age of twelve, 876 had seven or more carious teeth.

The foregoing figures relate to children attending the Poor Law Schools, workhouses, and reformatories. Equally unsatisfactory conditions have been shown to exist among children in better circumstances ; for example, 560 boys, average 13 years 7 months, belonging to one of our large public schools, had lost 701 of the permanent teeth, while 3,521 were carious. In the mouths of 87 per cent. of the boys caries was present. In

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[1] Memorandum in regard to the condition of the teeth of school children, published by the British Dental Association, p. 64, showing the relative ratio per hundred children having unsound teeth, defective deciduous teeth, and defective permanent teeth.

[2] The Seventh Annual Report of the School Medical Officer of the Salop County Council, 1914.

America, Germany, Hungary, and other countries, similar investigations have yielded much the same results. The investigations of Unghvari (Hungary) and Berten (Würzberg) show the percentages of carious teeth to be 87·2 and 83; while Röse, in an extended inquiry in Baden and Thuringia, and Fenchel (Hamburg) showed caries present in 98·75 per cent. and 96·4 per cent., respectively. The statistics of Röse and Fenchel indicate that, as regards caries, the teeth of English children compare favourably with those of German children.

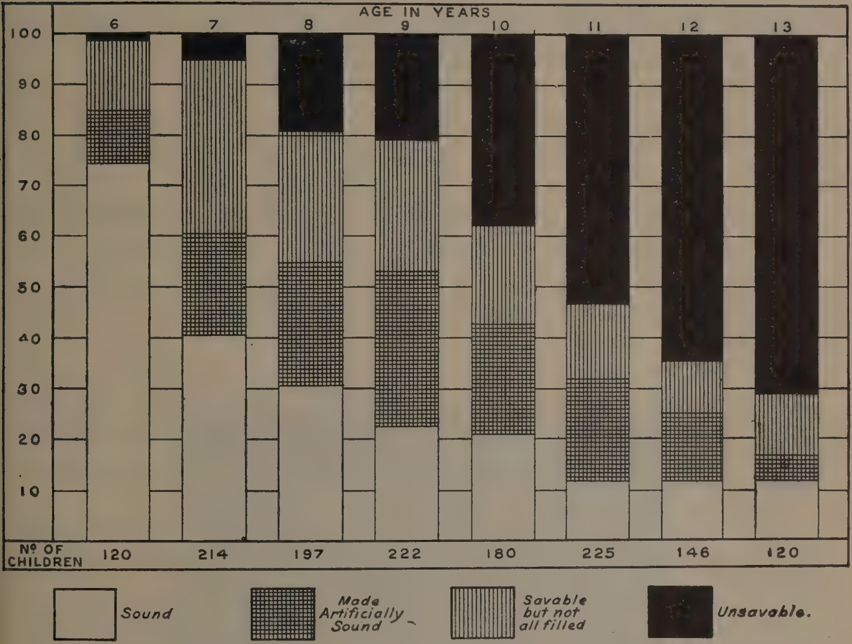

FIG. 397.—Diagram showing the relative liability to caries according to age.

(From *Brit. Dent. Journ.*, xxx, 572.)

The rapid increase in caries between the ages of six and thirteen years is well shown in the diagram (fig. 397) prepared by F. W. Richards.

The foregoing figures will suffice to illustrate the appalling ravages of dental caries among modern races. It is satisfactory to know that other investigations have brought forth results which show more hopeful conditions. For example, C. M. Cunningham [1] found that among fifty-four children in the Arran Islands there were thirty-three perfect dentures, and clinical experience undoubtedly shows that the number of perfect dentures is increasing among the children of the more intelligent sections of the community.

Recent reports of school dentists indicate, too, a welcome decline in dental caries. T. A. Edmondson found that of 2,403 children inspected in 1920, 15·7 per cent. had sound dentures in contrast to 10·8 per cent. in 1919.

(4) Animals.

Investigation by one of us (J. F. C.) [2] of the incidence and other relevant details of caries in animals brings to light the following facts :—

Caries occurs in wild, captive, and domestic animals, but least in the wild.

In wild animals it is more common to find caries starting in the cementum or dentine and spreading superficially, but in captive and domestic animals the initial lesion more often starts in a position where food debris may find lodgment, and penetrates the enamel and dentine as in man.

In wild animals its incidence varies considerably in different species, and in some species varies according to the locality from which the particular specimens come.

The primates are naturally of most interest to us, and of these : In the anthropoids more than 97 per cent. of all cavities were approximal, and over 76 per cent. in incisor teeth ; the incidence of caries in this genus varying from nothing in 88 specimens of the siamang (a species of gibbon), to 13 cavities in 5 specimens in a total of 689 gorillas, and 76 cavities in 21 specimens in a total of 465 chimpanzees (fig. 398).

Examination shows that the teeth had invariably been either exposed to considerable attrition leading to a small portion of enamel being flaked off and so giving rise to a stagnation area, or had the cement exposed by the packing of food in the interdental spaces.

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[1] *Brit. Dent. Journ.*, xviii, 652.

[2] ‘Variations and Diseases of the Teeth of Animals,’ 1936, Chap. XIX.

A comparison of the incidence of caries in wild and captive Old and New World monkeys as in the following tables is instructive and confirmatory of the generalisations already given :—

THE OLD AND NEW WORLD MONKEYS.

Family or Sub-Family.	From the Wild State.				From the Captive State.			
	No. of specimens.	No. showing caries.	No. of carious teeth.	Percentage of specimens showing caries.	No. of specimens.	No. showing caries.	No. of carious teeth.	Percentage of specimens showing caries.
Old World :								
Cercopithecinæ	2,785	78	274	2·8	924	75	223	8·1
Colobinæ	2,322	2	3	0·1	48	—	—	—
New World :								
Cebidæ	2,122	35	66	1·6	160	4	14	2·5
Hapalidæ	211	1	4	0·5	35	—	—	—

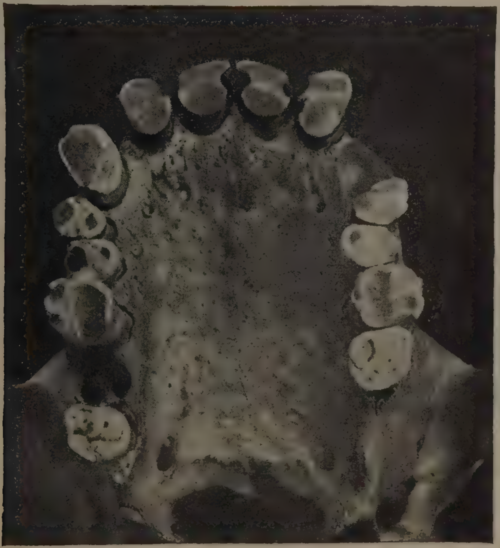

FIG. 398.—Anthropopithecus troglodytes (Chimpanzee). Maxillæ showing caries of the incisors.

From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

Of the Old World monkeys the following table shows that caries is commoner in certain genera than in others :—

OLD WORLD MONKEYS—CERCOPITHECINÆ GROUP.

Caries in the Different Genera.

Genus.	From the Wild State.				From the Captive State.			
	No. of specimens.	No. showing caries.	No. of carious teeth.	Percentage of specimens showing caries.	No. of specimens.	No. showing caries.	No. of carious teeth.	Percentage of specimens showing caries.
Cercopithecus	1,533	63	223	4·1	338	26	67	7·7
Erythrocebus	42	—	—	—	54	10	23	18·5
Cercocebus	277	4	15	1·4	38	1	4	2·6
Macaca	588	10	34	1·7	305	18	50	5·9
Papio	288	1	2	0·3	153	15	50	9·8
Mandrillus	36	—	—	—	27	5	29	18·5
Theropithecus	21	—	—	—	5	—	—	—

The starting-point of the cavities (in so far as it can be judged) in these Old World monkeys (figs. 399 and 400) differs considerably in wild and captive animals, as follows :—

	Wild State.	Captive State.
Approximal cavities	94·2	18·6
Crown cavities	5·8	81·4

and the distribution of these cavities (figs. 399 and 400) in the teeth affected varied as under :—

Teeth affected.	Wild State.	Captive State.
Incisors	73·3 per cent.	3·1 per cent.
Canines	—	0·9 ,, ,,
Premolars	11·7 ,, ,,	8·5 ,, ,,
Molars	15·0 ,, ,,	87·5 ,, ,,

Of other primates two of 650 wild lemurs showed caries, as compared with two of eighty-five captive lemurs.

Wild animals in other orders do not commonly have caries; of 2,687 ungulates only four showed lesions simulating caries, of 7,635 carnivores not more than six (fig. 401), the shape of the teeth and the nature of the food of carnivores when wild being doubtless protective; and 3,800 wild rodents showed no caries.

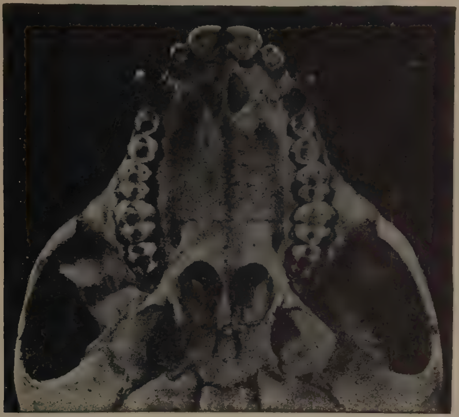

FIG. 399.—Cercopithecus mona grayi (Gray's Monkey). This specimen shows the formation of cavities in the first incisors.

(From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

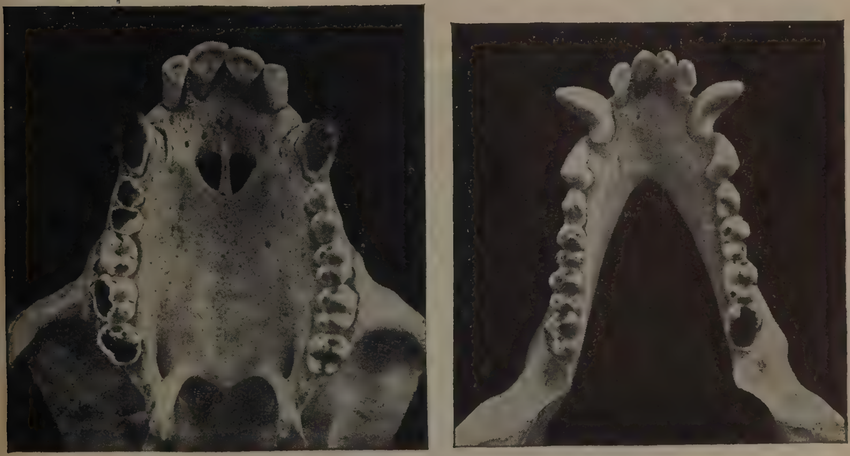

FIG. 400.—Cercopithecus, R.C.S. Odonto. Section G 40.31. A specimen showing extensive caries in a captive animal.

(From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

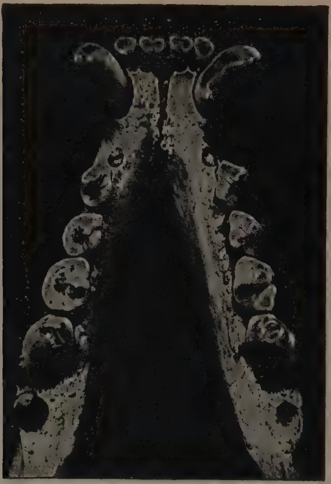

Fig. 401.—Paguma grayi (Himalayan Palm Civet). B.M. 14.7.10. 243. Mandible ; there are cavities in the posterior portions of the first molars. (From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

In domestic animals caries has been found in sixty-six of 484 London horses, involving as many as ten teeth in some cases (fig. 402). It is rare in dogs (fig. 403), occurring in four only of

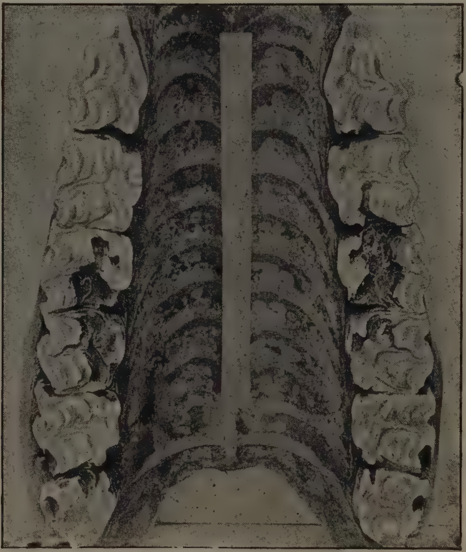

Fig. 402.—Maxillæ of a horse showing extensive caries of the teeth. (From *Trans. Odonto. Soc.*)

762 museum specimens, but has been known to occur in sheep and hutch-fed rabbits.

For further information on the comparative incidence of caries the student should consult the work of reference from which the above facts and tables are taken.

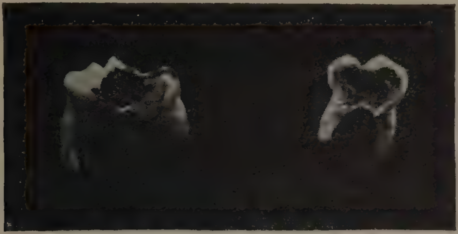

FIG. 403.—Canis familiaris (Domestic Dog). R.C.S. Osteo. Section G 139.3. Two carious molar teeth.

(From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

(B) FREQUENCY IN INDIVIDUAL TEETH.

The various groups of teeth in human beings are subject to caries in different degrees. The statistics bearing on this question are nearly all derived from records of extractions, and it is needless to say that statistics so obtained will not correctly represent the relative tendency to caries in the different groups. The permanent first molars are more subject to caries than other teeth, and the mandibular more than the maxillary. The permanent first molars are erupted early, and the liability to caries is due to the unhygienic conditions in the mouth at this period rather than to any inherent structural defect. The caries nearly always commences on the occluding surfaces. These teeth are probably less liable to decay on the approximal surfaces than the maxillary premolars. The permanent second molars probably follow next to the permanent first molars in their liability to caries, the mandibular being attacked more frequently than the maxillary.

In tabulating the incidence of caries in the four permanent first molars and the four maxillary incisors in the same mouths in four different groups[1] of children one obtained the following figures for comparison; several hundreds of children were tabulated in each group, and the figures worked out as for 100 children in each group.

---

[1] *Trans. School Dent. Soc.*, 1914, vii, 6. (E. S.)

Sex.	Average age.	Carious permanent 1st molars.	Carious maxillary incisors.
Male	12·58	190	18
Female	12·65	170	4
Male	12·55	234	22
Female	12·6	277	32

It is extremely difficult without trustworthy statistics to place the premolars correctly as regards their liability to caries.

Experience affords little evidence on which to choose between the maxillary first and second premolars in this respect, but with regard to the mandibular premolars the liability to caries is more marked in the second than in the first. The mandibular incisors are comparatively immune to caries, and this is due as much to their shape as to the constant mechanical cleansing action of the tongue and lips. The liability of the third molars to caries in mouths where all the teeth are present is attributable to difficulty in keeping them free from the lodgment of food debris.

Frequency in Relation to Sex and Age.—It is generally admitted that caries is more common in females than in males. The observation of Röse[1] on 6,280 children, boys and girls being about equally divided, showed the percentage of caries in boys to be 26 and girls 26·2. Caries is more active during the period of growth of the individual than subsequently. A fresh period of activity often appears with conditions which lead to exposure of the roots of the teeth, the caries commencing in the cementum (see also p. 358).

(C) MORBID ANATOMY AND PATHOLOGY.

(1) MACROSCOPICAL APPEARANCES.

(*a*) **Enamel.**—In the enamel, the naked-eye appearances of caries can best be observed upon the approximal side of a molar or premolar. The first indication of the presence of caries is that the enamel loses its normal polish and translucency. Next, a whitish spot appears which gradually becomes darker. When the process is slow, the discoloration is more marked than when the process is rapid. As the interprismatic substance becomes disorganised the prisms lose their support and, breaking away, are mechanically washed away and a cavity is left which varies somewhat in form, being sometimes broad and shallow with indistinguishable margins, at other times deep with sharp rugged margins. The destruction advances until the dentine is reached, when it spreads along the amelo-dentinal junction.

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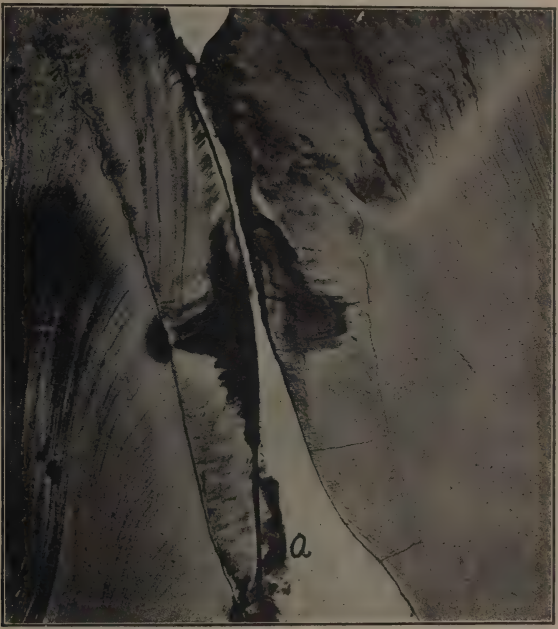
[1] Röse: 'On the Decay of Teeth in the National Schools of Germany,' *Oesterreich-Ungarische Vierteljahrsschrift für Zahnheilkunde*, translated in Ash's *Quarterly Circular*, March and June 1895.

At times, decalcification may have attacked the dentine before any disintegration of the enamel has occurred (see fig. 404).



× 35.

FIG. 404.—Decalcification of the enamel without loss of substance (Miller). (From *Dental Cosmos.*)

When caries commences on the approximal aspects of the teeth, the actual point of contact is not always decalcified, but around it is a zone showing decalcification. This zone corresponds to the area on the tooth where fluid would be held by capillary attraction.

When caries which has started on the approximal surface reaches the under surface of the enamel (as is frequently the case in molars and premolars), the enamel at that part appears bluish-white and translucent, especially by artificial light. This appearance is produced by the decalcified tissue which has not been removed by the saliva. As the caries reaches the surface, the enamel grows whiter, and eventually becomes so weak that mastication fractures it, and the under surface is found to be of a soft, cheesy consistency. Caries commencing on the dentinal aspect of the enamel and proceeding to the surface is known as 'secondary enamel decay' (figs. 405 and 406).

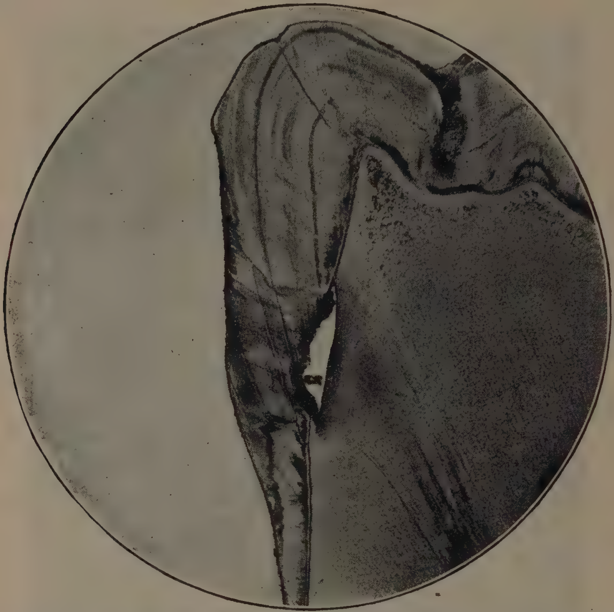
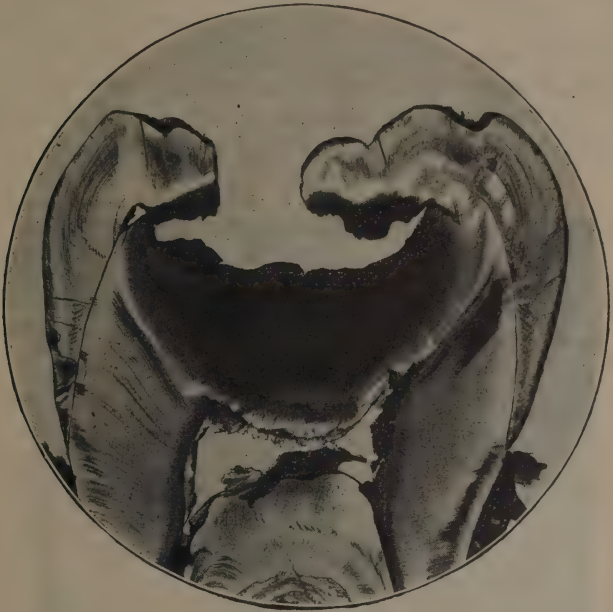


Fig. 405.—Primary and secondary caries of enamel, showing marked difference in their character (Miller).

(From *Dental Cosmos.*)

In children in whom rickets develops towards the end of the first year and after the eruption of the deciduous incisors, these teeth are often completely decalcified, brown, leathery masses of dentine being left maintaining the contour of the teeth. There is no proteolytic dissolution of the colloidal substance. In these children the saliva is quite acid.

(*b*) **Dentine.**—The appearances of caries in dentine differ considerably from those in enamel. The dentine becomes of a tough, cartilaginous consistence, and not soft and cheesy as with the enamel. After softening, the tissue undergoes disintegration, and a cavity is formed. Pigmentation accompanies the process and, as in caries of the enamel, the discoloration depends to a great extent upon the rate of progress of the disease.



× 10.

FIG. 406.—Secondary caries of enamel. The acid destroys the enamel as it advances. Apparent increased transparency of the enamel (Miller).
(From *Dental Cosmos.*)

Caries in dentine may spread in any direction and with varying degrees of rapidity. The rapidity of its progress depends upon the intensity of action of the various ferments and the chemical and physical structure of the dentine. The direction taken is largely determined by structure.

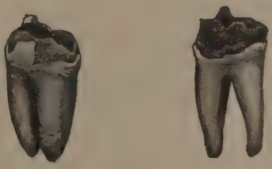


FIG. 407.

In well-calcified dentine decay will extend in the direction of the pulp rather than laterally, and may on occasion involve only a restricted zone of dentine fibrils; this is known as 'penetrating caries.' In dentine poorly calcified decay will extend laterally and considerably undermine the enamel; this is particularly seen when hypoplasic teeth become carious, when an appearance as seen in fig. 407 is often produced.

In '**rapid caries**' the dentine can be removed in large leathery masses, the action of the acid being more rapid than that of the peptonising organisms. This condition is called '**caries humida.**' When the carious process is slow, the action of the peptonising organisms almost keeps pace with the action of the acids.

(*c*) **Cementum.**—Caries of the cementum is less common than caries of the dentine or of the enamel. It generally starts at the neck of the tooth, though it may attack any part of the root when the roots are exposed through loss of the periodontal membrane. The cemental tissue first becomes softened, and disintegration follows, leading to the formation of shallow cavities. The cavities become widely extended, but owing to there being no circumscribed points of retention, they are seldom deep. An exception, however, occurs at the angle formed by two roots of a molar, the cavity at this point often becoming deep.

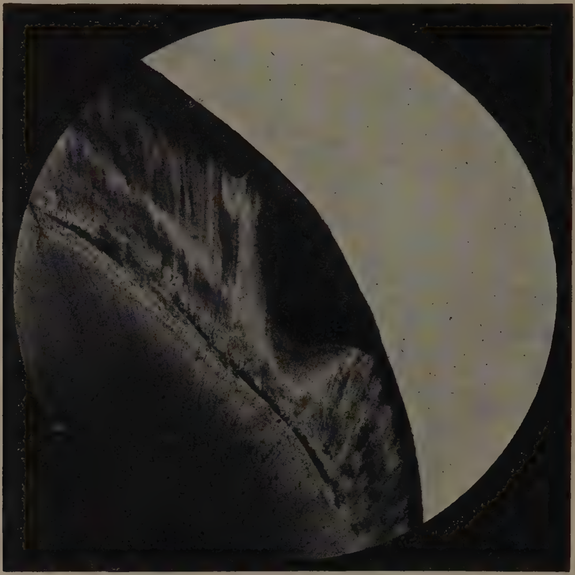


FIG. 408. (J. Howard Mummery.) (From *Brit. Dent. Journ.*)

(*d*) **Nasmyth's Membrane.**—The naked-eye appearance of destruction of Nasmyth's membrane is a more or less pronounced discoloration.

(*e*) **Reactions and Phenomena accompanying caries :—**

(i) *Enamel.*—J. Howard Mummery, in 1926,[1] first described and figured a reaction in the enamel against the advance of caries; this was shown as a zone of hypercalcification deep to superficial caries in enamel (fig. 408). Confirmation of the possibility of this occurring is given by one's work on enamel [2] showing also that it may react in a similar manner to traumatic injury; this also appears to demonstrate that the hypercalcification takes place via the interprismatic substance, and that the prisms become hypercalcified as well.

The fact that the tissue at this point is denser is shown by it being less permeable to staining influences than is normal enamel, and so far as we know increased density can only be attained by an increase in calcification.

(ii) *Dentine.*—In the dentine a reaction of a similar nature takes place; it is more readily seen in slow caries. A translucent zone, less, or even quite, impermeable to stains appears between advancing caries and the tooth pulp; in shape it assumes the area of a cone with its apex towards the pulp; the dentine on either side of the zone may be freely permeable (fig. 409). As caries advances this zone of hypercalcification extends to the tooth pulp, which, to protect itself further, then forms secondary dentine in continuity with the ends of the affected tubules. That this reaction is a vital one is confirmed by its occurrence only in teeth having living pulps and its absence when caries is artificially produced in the laboratory. That it is due to hypercalcification is known from the fact that it becomes progressively less permeable and ultimately impermeable to stains; as this calcification of the tube contents takes place, the refractive indices of the tube contents and dentine matrix thereby become approximated so that translucency of greater or less degree is produced. If this zone were produced by decalcification the portion of dentine it involved would be more readily stainable than the other dentine of the tooth, and the zone would extend in all directions around the carious focus, but neither is the case.

It has been stated that the translucent zone area in dentine, whether produced as a reaction to the advance of caries or to the advance of physiological attrition, is an area of ' dead ' tissue;

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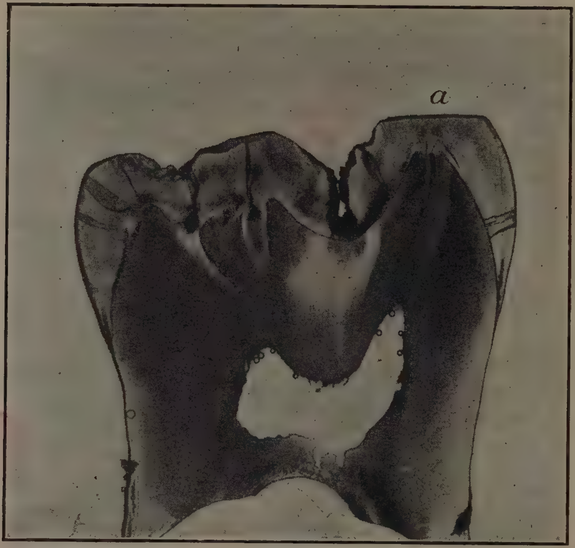
[1] *Brit. Dent. Journ.*, 1926, xlvii, 473.

[2] *Proc. Roy. Soc. B.*, 1930, 106, 376. (E. S.)

while there is no conclusive evidence in favour of this contention, there is evidence, apart from that already given as to its vital character, that it may be living ; dentine exposed by attrition is often very sensitive to tactile and acid stimuli. The possibility of the zones being ‘ dead ’ is based on the statement that if they are impermeable to stains they must also be impermeable to vital influences, and so only present as inert chemical substances ; but nine-tenths of the body is water, itself an inert chemical, and chemists can account for 100 per cent. of the body purely as chemicals, and all chemical substances as such are inert. In addition, the formation of secondary dentine attached to and in organic continuity with the translucent zone is, by comparison, very strong evidence that such zones are living, as regeneration (such as the formation of secondary dentine) can only take place in continuity of structure between live tissues, and such continuity between secondary dentine and the translucent zone exists physically and can be seen as organic continuity microscopically. These zones may also be found under intact enamel which has not been subjected to attrition.



× 10.

FIG. 409.—Section of molar showing typical translucent zone in the dentine (Miller). (From *Dental Cosmos.*)

(iii) *Pigmentation.*—The pigmentation accompanying caries varies from a pale yellow to black; the more rapid the process, as a rule, the lighter the colour. As the discoloration in carious teeth is produced solely by outside agency and is also seen in dentine free from caries, it may be safely assumed that it is merely an incidental phenomenon of the disease and not an active agent in producing the disease. The discoloration is in all probability produced by colouring matter derived from such things as tea, tobacco, etc.; it is improbable that it is produced by chromogenic bacteria, because dentine exposed by attrition is sometimes similarly superficially stained, and bacteria would not have the opportunity to remain on such surfaces to produce pigment.

(2) CHEMICAL CHANGES.

The chemical changes which take place in carious teeth consist in a decrease in the amount of lime salts, with loss also of organic matter. In analyses undertaken by Miller it was found that carious dentine contained only about one-thirteenth of its original amount of lime salts, while the organic material was also reduced by two-fifths.

(3) MICROSCOPICAL APPEARANCES.

(*a*) **Enamel.**—The microscopical appearances have been shown by C. F. Bödecker[1] and H. C. Malleson[2] in microtome cut sections. In carious conditions these show that a bacterial plaque is formed on the surface of the enamel, which produces an opacity by acting on the interprismatic substance; infection then proceeds either along the space previously occupied by the interprismatic substance or along a fissure which may be developmental or otherwise, the progress of caries being more rapid when a fissure is present. As the interprismatic substance is dissolved the prisms lose their support and break away, leaving a cavity (figs. 410 to 412). Considerable importance was attached to the presence of bacterial plaques by Leon Williams and G. V. Black, who had previously described them.

(*b*) **Dentine.**—If longitudinal sections of carious dentine which have been stained to demonstrate micro-organisms be examined microscopically, the following points will be observed.

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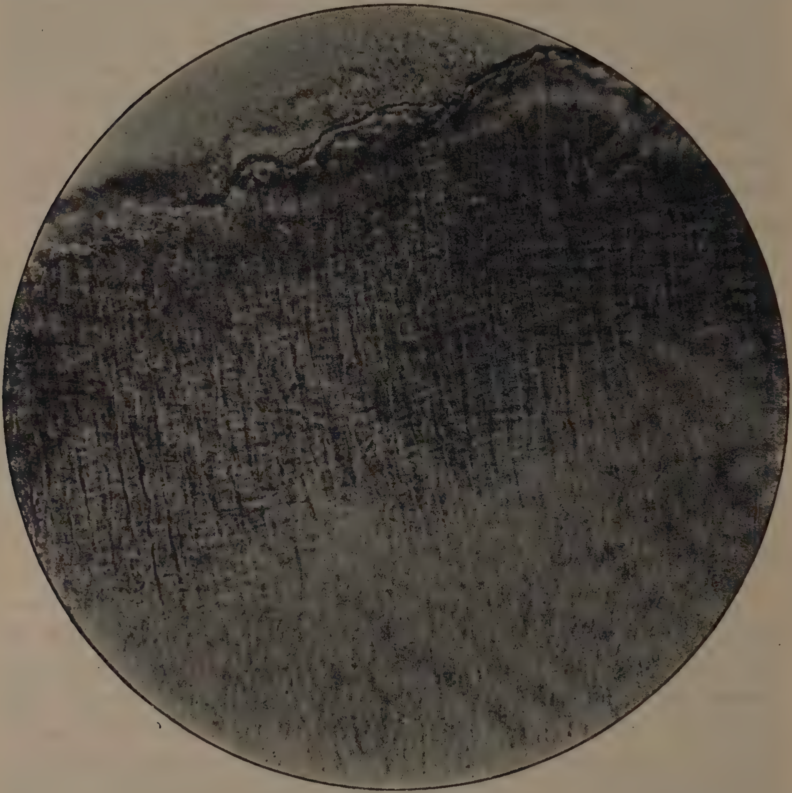
[1] *Dental Review*, May 1905 and April 1906.

[2] *Brit. Dent. Journ.*, Aug. 1925.

Bordering the surface (fig. 413) the dentine is hollowed out in an irregular manner, the cavities presenting no definite shape (*a*). A little deeper in the substance of the dentine irregular masses of stain can be detected (*b*), and, in places, the stain presents a globular appearance (*c*). Lower down, streaks of stain are to be seen (*d*), while still farther down the dentine is unstained (*e*).



× 800.

FIG. 410.—Showing commencement of caries. Enamel rods separated by solution of cement substance and discoloured by action of acid. (Leon Williams.)

The interpretation of these appearances is as follows :—

(i) The unstained part (*e*) is dentine in a decalcified but uninfected condition.

(ii) The streaks of stain (*d*) are micro-organisms occupying individual dentinal tubes.

(iii) The globular masses are micro-organisms occupying the tubes and adjacent dentine matrix ; in other words, the tube

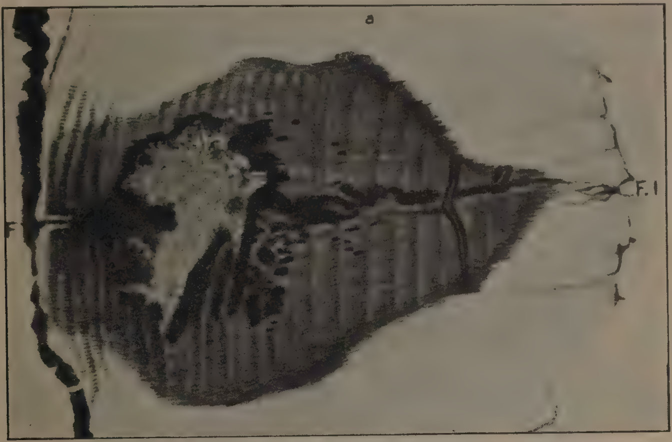


Fig. 411.—Enamel from the distal surface of a premolar. Stained Heidenhain's hæmatoxylin. Showing cavity filled with micro-organisms, and changes in the surrounding enamel. (*b*) (F.F.I.) Infected fissure, (*a*) normal enamel. ×80. (H. C. Malleson.) (From *Brit. Dent. Journ.*)

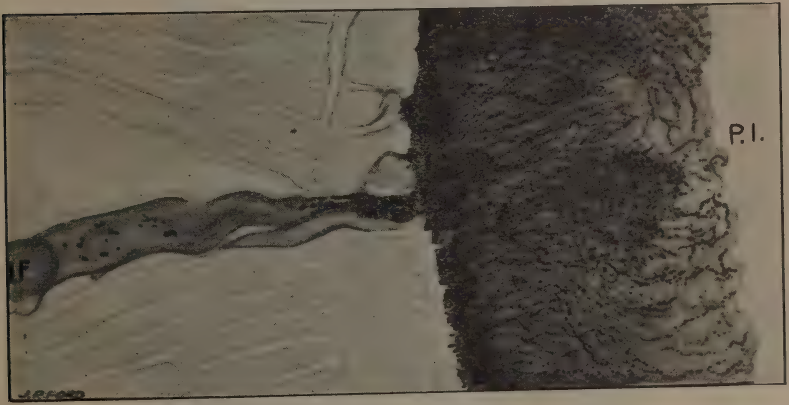
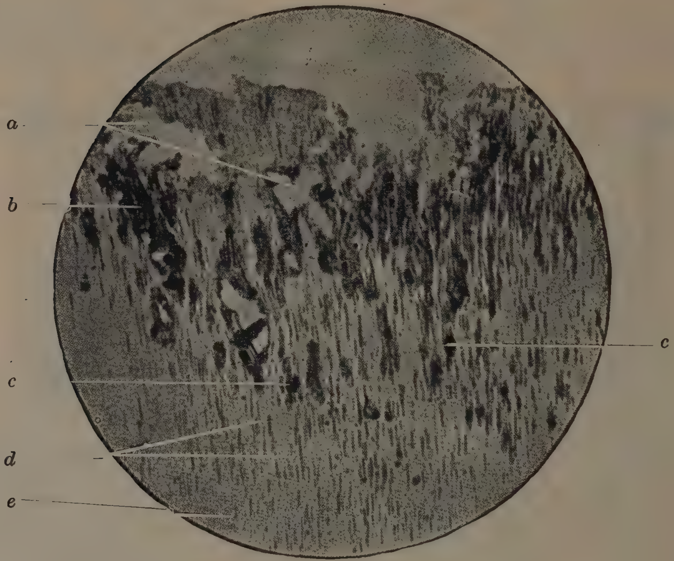


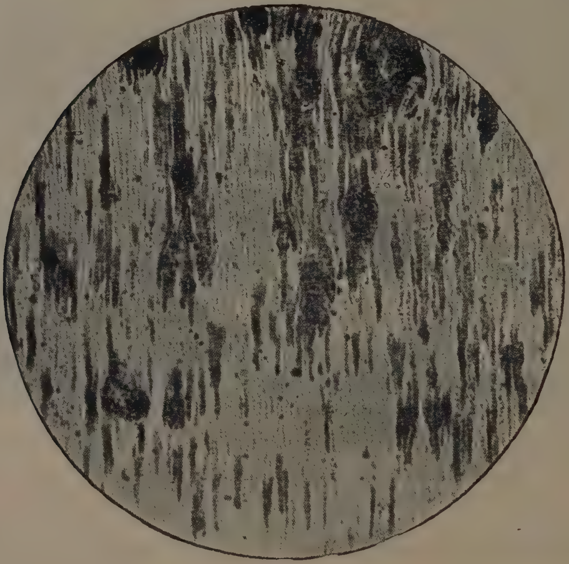
Fig. 412.—Enamel from the approximal surface of a young premolar. Stained Gram and Eosin, showing bacterial plaque and infected fissure. × 800. (H. C. Malleson.) (From *Brit. Dent. Journ.*)

sheaths and dentine matrix have disappeared at those points and micro-organisms have taken their place. These globular masses are termed '**liquefaction foci**' (fig. 414).



× 80.

FIG. 413.—Longitudinal section of carious dentine. (A. Pringle.)



× 150.

FIG. 414.—Longitudinal section of carious dentine, showing liquefaction foci. (A. Pringle.)

(iv) The irregular masses are formed by the fusion of 'liquefaction foci.'

In sections containing interglobular spaces, the spaces are generally seen to be filled with masses of organisms. Figs. 415 to 417 are longitudinal sections through carious dentine stained to demonstrate the micro-organisms.

If sections of carious dentine are stained to demonstrate micro-organisms only, the softened parts towards the cavity are stained, and towards the normal dentine unstained, which clearly shows that softening precedes infection. Miller has termed the unstained portion the '**non-infected zone.**' The micro-organisms show a greater tendency to spread towards the pulp than in a lateral direction, but this is not always the case especially when a large number of interglobular spaces are present. The dividing line between the infected and the non-infected zones is often well marked (fig. 418).

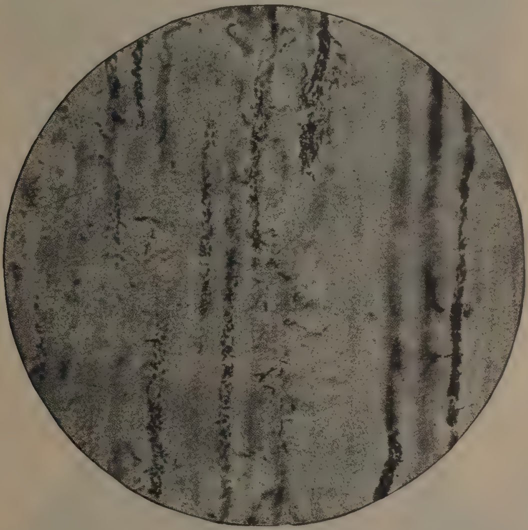
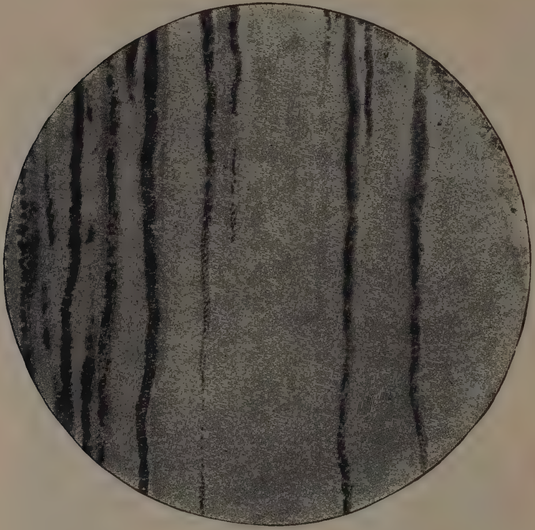

× 650.

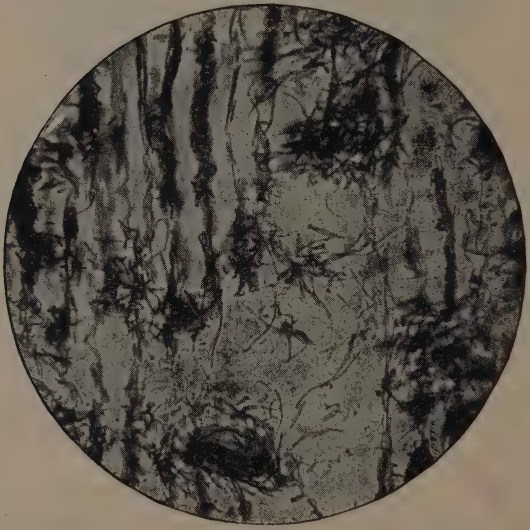
Fig. 415.—Longitudinal section of carious dentine, showing rod-shaped organisms in tubes. (J. Howard Mummery.)

In addition to micro-organisms, *rod-shaped fragments or elements can usually be demonstrated in the tubules* and are also seen in artificially produced caries. These elements, the source of which is not definitely known, are conjectured to be either



× 650.

FIG. 416.—Longitudinal section of carious dentine, showing tubes filled with micrococci. (J. Howard Mummery.)



× 650.

FIG. 417.—Longitudinal section of carious dentine, showing tubes filled with organisms. (J. Howard Mummery.)

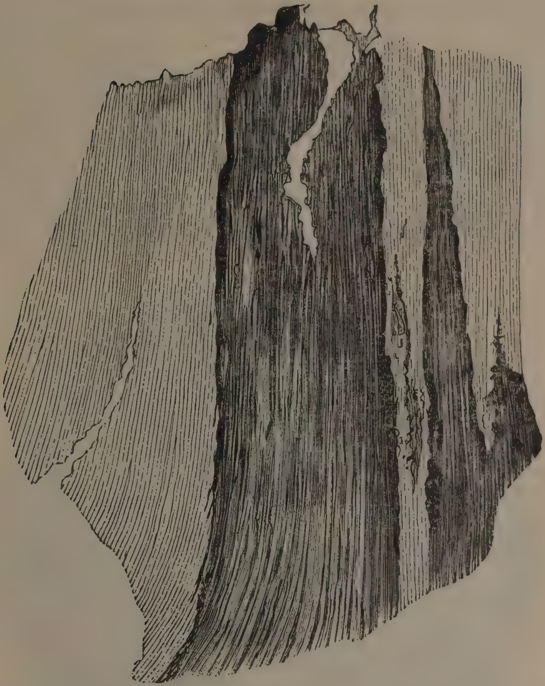
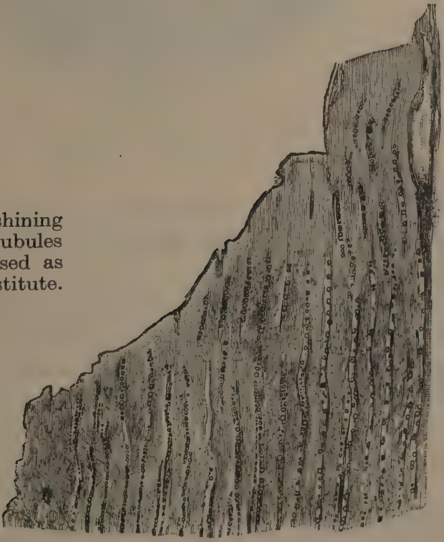


FIG. 418.—Carious dentine, showing that, laterally, the boundary between the infected and non-infected parts may be very regular. (Miller.)

FIG. 419.—Row of shining granules in the tubules from a tooth used as an artificial substitute. (Miller.)



(i) portions of consolidated fibrils; (ii) pieces of the sheaths of Neumann; or (iii) casts of the enlarged fibrils. Miller finds that these elements disappear immediately they are brought into contact with inorganic acids (dilute sulphuric acid), but become more distinct when organic acids are used, and he therefore thinks it probable that they are lime formations. *Rows of shining irregular granules* are also met with generally in the area in advance of caries (fig. 419). It is possible that these granules have the same origin as the rod-shaped elements, for, if the latter be crushed, granules can be produced.

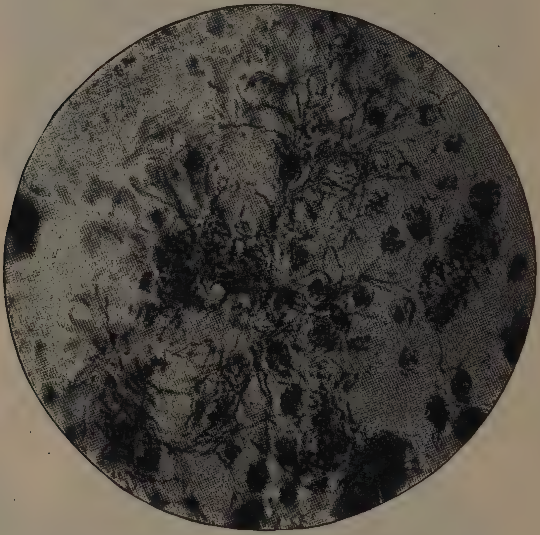

× 650.

FIG. 420.—Transverse section of carious dentine, showing enlarged tubes filled with micro-organisms.

Transverse sections near the carious cavity show the tubes enlarged and filled with micro-organisms (fig. 420). In transverse sections taken nearer to the normal dentine, it is found that the sheath of the dentinal tubes is considerably enlarged, owing, probably, to decalcification. The appearance of the dentine in this condition has been termed the *tobacco-pipe stem* appearance (Tomes). This is shown in fig. 421.

The tobacco-pipe stem appearance is also seen in caries occurring in teeth used as substitutes, and in caries produced by artificial means.

In the *Trans. Odonto. Soc.*, xxiv, 90. C. S. Tomes has figured a tooth found in the graveyard of an old church. The cementum and dentine of this tooth have been bored in many directions by a fungus, probably the *Saccharomyces mycoderma* (Miller). The penetration of the dentine by *Saccharomyces mycoderma* is, according to Miller, frequently seen in teeth used as artificial substitutes. It is not at all improbable that other fungi may possess the power of penetrating dentine.

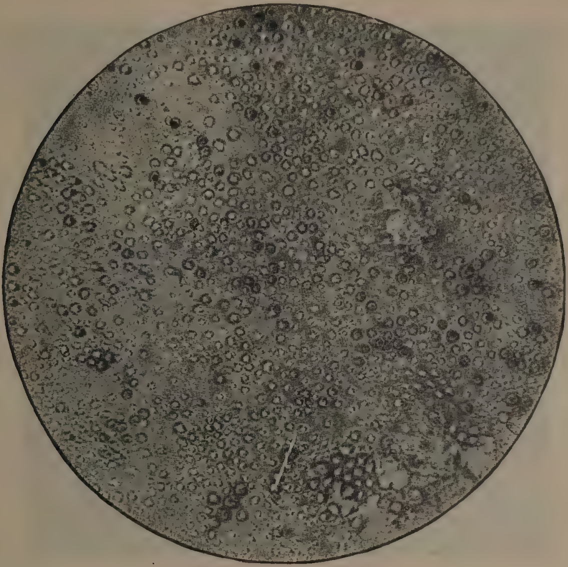

× 200.

FIG. 421. (A. Pringle.)

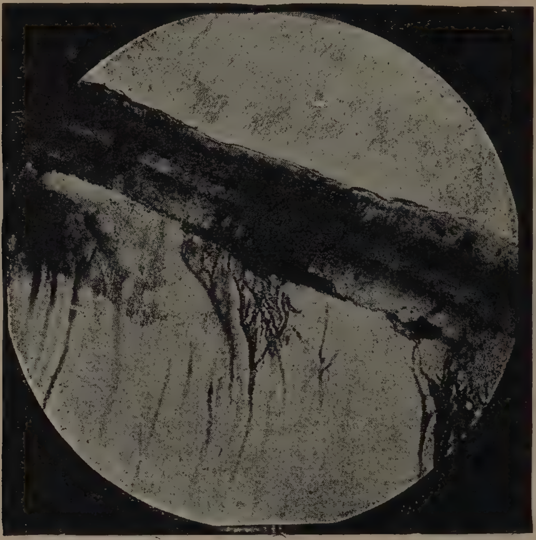
F. J. Bennett has shown teeth from graves in Central America, with the dentine almost completely destroyed by a fungus.

(*c*) **Cementum.**—Caries of cementum (fig. 422) resembles caries of dentine in some details, especially when the micro-organisms penetrate, as they commonly do, along the line of Sharpey's fibres.

There is some reason to doubt whether the destruction of the cementum is always brought about by the same process as destruction of the enamel. There are individuals who are free from caries during the greater part of their lives, but who eventually become susceptible to caries of the cementum.

When cementum is exposed, the outer layer is liable to be robbed of nutrition and die or to be considerably lowered in vitality. In such tissue autolytic changes may take place, and it is possible that proteolytic enzymes formed from proteid residues or mucus may be responsible for the primary decay in this tissue.

In practice one meets with cases in which the roots of the teeth have been exposed by abrasion, and there is a patch of slightly softened dentine in a position away from the risk of food lodgment. In many of these cases there is an absence of caries of enamel. It is difficult to account for this softening of the cementum on the lines of carbohydrate stagnation, and it would seem that other agencies may be at work in the production of this condition. In some instances the cavities in the cementum are apparently the result of direct absorption of tissue associated with an inflammatory condition of the gum margin.



× 600.

Fig. 422.—Caries in cement. (Preparation by Miller. Photomicrograph by J. Howard Mummery.) (From *Brit. Dent. Journ.*)

G. Preiswerk,[1] as a result of experiments, came to the conclusion that caries could occur in an alkaline reaction. The organic ground substance is first destroyed, the disintegration being brought about by proteolytic action, and with the dissolution of the connective tissue the calcium salts disappear. It is possible that the slow destruction of roots, so often seen in the mouth, is brought about by this means and not by acids formed from carbohydrate debris. With the disappearance of the cementum, a cavity in time forms which allows of the retention of food debris, and the process may then follow that seen in caries elsewhere.

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[1] 'Atlas and Text-book of Dentistry,' 1907.

(*d*) **The Effect of Caries on the Pulp.**—This portion of the subject is dealt with in detail in a later chapter (XIII) treating of the pathology of the dental pulp.

(4) BACTERIOLOGY.

The first systematic account of the action of micro-organisms in caries was published by Leber and Rottenstein in 1867. These authors described caries as due partly to the action of acids and partly to the proliferation in the tubes of the dentine of a definite micro-organism, the *Leptothrix buccalis* ; they also expressed the view that the growth of the fungus in the substance of the dentine could not take place without a preliminary decalcification of the tissues of the teeth by acid. The acid they believed was formed by the fermentation of the particles of food lodged between the teeth and in fissures on the enamel, and considered it probable that lactic acid was formed in this fermentation.

In 1881 Underwood and Milles [1] showed that caries was absolutely dependent upon the presence and proliferation of micro-organisms. At a later date Miller established the facts that some organisms had the power to produce acids from foodstuffs in sufficient quantity to decalcify tooth structure ; others the power to break down decalcified tissue ; while others possessed both properties. In 1883 Miller [2] isolated four types of bacteria from carious dentine ; five years later, Vignal and Galippe [3] examined eighteen carious teeth, in all of which they demonstrated four types of bacteria, a fifth type was found eight times, and a sixth type five times. K. W. Goadby [4] isolated eighteen types of organisms and divided them into two groups: (*a*) Acid-forming bacteria or bacteria capable of producing acids by the fermentation of carbohydrates; (*b*) liquefying bacteria which, either by their own action or by the production of proteolytic enzymes, cause digestion of the decalcified dentine.

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[1] *Trans. Internat. Med. Congress, London,* 1881, iii, 523.

[2] *Independent Practitioner,* July 1884.

[3] *L'Odontologie,* March 1889.

[4] 'The Mycology of the Mouth.'

Goadby has also shown that the number and varieties of bacteria on the surface of a carious cavity are more numerous than in the deeper layers; the varieties on the surface of the carious dentine are mostly aerobic liquefiers—many of them liquefy gelatin with great rapidity—and are capable of coagulating blood serum. Most of the organisms which liquefy coagulated blood serum will digest decalcified dentine. A small fraction were anaerobic liquefiers. The organisms from the deeper layers of carious dentine are rarely liquefying bacteria, but nearly all of them are capable of the production of acid from carbohydrates, especially glucose, maltose, and lactose. The organisms on the surface of carious dentine often possess the power of discolouring the medium upon which they are originally grown.

Goadby states that not all organisms capable of liquefying nutrient gelatin also possess the power of digesting the decalcified tooth cartilage or chondrinogen. From the deeper layer of the carious dentine he has frequently isolated an organism for which he suggests the name *Bacillus necrodentalis*, and he seems to think that this organism may stand in some causal relationship to caries, inasmuch as its characteristics fit in with the process of decay. In sixteen out of twenty cases he obtained the *S. brevis*, and as this organism and the *B. necrodentalis* are facultative anaerobes and are both acid producers, he considers that they may be largely instrumental in decalcifying the matrix prior to its disintegration by other organisms.

Another organism, hitherto unrecognised in the mouth, has been found by Goadby constantly associated with organisms from the surface of carious dentine, and in two out of twenty cases it was associated with organisms from the deeper layers. This organism, he considers, belongs to Streptothricæ, and has been termed by him the *Streptothrix buccalis*.

In the *British Journal of Experimental Pathology* (June 1922), McIntosh, James, and Lazarus-Barlow described two organisms which they isolated from carious dentine, the *Bacillus acidophilus odontolyticus* I and II.

The deeper layers of carious dentine were emulsified in acid broths and, after twenty-four hours' incubation, broths of varying degrees of acidity were heavily inoculated and, from these, agar plates were inoculated after twenty-four to forty-eight hours.

'The organisms isolated by the above method fall, morphologically, into two main groups: (*a*) Type I, a long, thin bacillus, $0 \cdot 75 \times 2–3\ \mu$, which occur singly, in pairs or chains, having a marked tendency to parallelism or palisade formation in dried films (fig. 423); and (*b*) Type II, a shorter bacillus, $0 \cdot 75 \times 1–2\ \mu$, usually occurring in chains (fig. 424). After prolonged subculture on agar, Type I fails to form chains. Both organisms are non-motile and Gram-positive; both are aerobes and facultative anaerobes. In broth there is in most cases uniform turbidity, but in a few cases the growth settles at the bottom. On ordinary agar the colonies may appear as minute points after twenty-four hours' incubation, but often do not become visible till after forty-eight hours; they are small, round, greyish and opaque, with a finely granular appearance under the low power and a regular outline. In size, the colonies are about 0·5–1 mm. in diameter; on serum agar, rather larger, up to 2 mm. In a gelatin-agar shake culture, the colonies have a rough biconvex

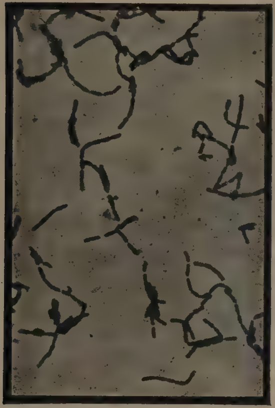

FIG. 423.—Type I bacillus: smear preparation from agar slope culture, showing palisade and chain formation. Gram. × 1,000. (From *Brit. Journ. of Experimental Pathology.*)

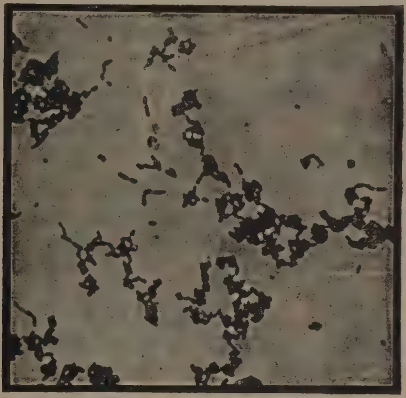

FIG. 424.—Type II bacillus: smear preparation from agar slope culture. Gram. × 1,000. (From *Brit. Journ. of Experimental Pathology.*)

appearance which might be described as being “ Tam o’ Shanter ” shaped.’

Type I was isolated in pure culture from thirty-eight and Type II from eighteen of the fifty teeth examined.

‘ As regards the fermentation of sugars, of the thirty-eight strains of Type I isolated thirty-three formed acid and no gas in glucose and lactose, no acid or gas in saccharose, and acid and clot in milk. The milk usually became acid on the second day and was firmly clotted on the third, with all except the top quarter to half an inch completely decolorised. Four of the remaining five strains formed acid in saccharose and glucose, but differed in their reactions in lactose and milk ; one strain formed acid in glucose alone. The action of Type II on sugars is nothing like so constant ; seven out of the eighteen strains isolated formed acid and no gas in glucose, lactose, and saccharose, and acid and clot in milk, the effect on the last-named being similar to that of Type I. Three strains formed no clot in milk after prolonged incubation, and two strains had no effect at all on milk. The remaining six strains varied considerably in their reactions.’

‘ In no case was gelatin liquefied by either type, nor was indol formed ; twenty-six strains were also inoculated into dextrin and dulcite without the formation of acid or gas.’

The authors have named the organisms *B. acidophilus odontolyticus* I and II.

Mouths of persons who suffered from caries were examined, and cultures of Type I were isolated in 30 per cent. of the cases and Type II in 20 per cent. (See also section on ‘ Experimental Reproduction of Caries,’ p. 342.)

There is no doubt that these organisms can cause the initial decalcification in dental caries. By putting teeth into emulsions of these organisms the complete phenomena of caries were artificially produced in that, not only did they produce the decalcifying acid but, when deprived of carbohydrates, they had a proteolytic action as well. Though these organisms can live in a fairly high concentration of acid they grow more freely when the medium is slightly alkaline.

Kilian Clarke has isolated an organism, the *Streptococcus mutans*, which he states is responsible for the initial decalcification of enamel. As its name implies, the appearance of this organism is variable, and depends on the reaction of the medium in which it is grown; in an alkaline medium it appears as a non-capsulated coccus 0·75 μ in diameter growing in chains, in an acid medium as a bacillus 1·5 μ to 3 μ in length. It is Gram-positive, an aerobe and facultative anaerobe. The optimum reaction for its growth is *p*H 7.

Sterilised normal teeth were placed in glucose broth *p*H 7 cultures of this organism. The medium was changed daily so as not to keep the teeth too long in an acid solution; the enamel became considerably decalcified in from seven to thirteen weeks. If the process were allowed to continue for a longer period the dentine tubes became infected as in dental caries. The growth adhered to the surface of the enamel. Kilian Clarke affirmed that the earliest lesions of the enamel were caused by this organism which was always present in caries of the enamel; he maintained that the *Bacillus acidophilus odontolyticus* is not found in the earliest lesions.

In conclusion, it must be remembered that in the mouth caries is probably never produced by any one organism, and it is likely that other organisms, in addition to those specifically mentioned, may take part in and assist or inhibit the initial production of acid. Adami[1] has directed attention to the fact that organisms, in adapting themselves to altered environment, may alter their reactions, and it is very probable that this occurs in such mixed infections as occur in carious cavities.

(5) THE SOURCE OF THE ACID.

Foods consist of proteins, fats, carbohydrates, water, and salts. If *proteins* should stagnate in the mouth, but little acid reaction is produced, whereas alkaline products are produced in excess, particularly in the later stages, so that the general reaction is alkaline. This cannot directly affect enamel. *Fats* are passed through the mouth practically unchanged and so cannot directly affect dental conditions.

Water is neutral and therefore inert.

Salts, derived mainly from vegetable foods, are mainly neutral, but even if not, the mastication of the vegetable fibre from which they are expressed prevents their remaining in contact with the teeth.

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[1] 'The Principles of Pathology,' 101.

Except that some of the above foods are cooked, the form in which they are taken differs but little from that in which wild animals may obtain them, and although dental caries is not unknown among wild animals it is relatively rare.

Carbohydrates.—Carbohydrates, when acted on by micro-organisms, may give rise to acid (lactic, malic, or succinic) ; it is the only food which can do so, and it is the only food which civilised man takes in a form markedly different from that in which any wild animal can take it. No wild animal can obtain carbohydrate in a pure form, either as starch or sugar ; as they obtain it it is always in combination with a fibrous or other ingredient (such as honey in the comb) which, when the article is masticated, tends to dislodge solid particles or fluids in solution from the crevices and interstices of the teeth ; particularly is this so towards the end of the act of mastication in the effort to extract the last remnants of 'goodness' from the article masticated ; besides, additional and free salivary flow is thereby occasioned. Under such normal circumstances it is probable that the salivary starch enzyme ptyalin may efficiently deal with what little, if any, starchy residue there may be.

Civilised man, on the contrary, takes most of his carbohydrate in an excessively refined and pure form, such as may readily adhere to or lodge around teeth ; moreover, some of the sugars he takes are in a more readily fermentable form than occurs pure in nature.

Carbohydrates occur in foods as Polysaccharides $(C_6H_{10}O_5)_n$, Disaccharides $(C_{12}H_{22}O_{11})$, and Monosaccharides $(C_6H_{12}O_6)$.

The Polysaccharides are cellulose, starch, and dextrin.

Cellulose.—This, the chief constituent of vegetable fibre and the vegetable cell wall, is probably not acted on by bacteria while in the mouth and so passes through it chemically unchanged.

Starch (cooked), if remaining in the mouth may be acted on by the ptyalin and converted into the disaccharide maltose and thence by a hydrolytic bacterial ferment called invertin into the monosaccharide dextrose ; bacterial action on monosaccharides can produce acid.

Uncooked starch, if the cellulose covering of the starch granules is intact, is protected from bacterial or enzyme action while in the mouth.

Dextrin is a soluble gummy substance derived from starch, concerning which but little is known of the action of bacteria on it.

Sugar.—Sugar is taken in two chemical forms :—

(i) Non-fermentable disaccharide ($C_{12}H_{22}O_{11}$) comprising cane and beet sugars, maltose, and lactose.

The non-fermentable disaccharides when in the mouth may be acted on by a hydrolytic bacterial ferment called invertin and converted into such monosaccharides as dextrose and levulose by hydrarisation thus :

$$\underset{\text{(cane sugar)}}{C_{12}H_{22}O_{11}} + H_2O = \underset{\text{(dextrose)}}{C_6H_{12}O_6} + \underset{\text{(levulose)}}{C_6H_{12}O_6}$$

(ii) Fermentable monosaccharide ($C_6H_{12}O_6$) comprising glucose, dextrose, levulose, galactose, grape sugar.

The fermentable monosaccharides can be acted on directly by bacteria to produce acid.

The acids which initiate dental caries by decalcifying the enamel have been thought by different authorities to be lactic, malic, and succinic acids, but principally lactic.

There is some difference of opinion as to whether the solid carbohydrates (starches) or the soluble (sugars) do the greater harm in the production of acid. The pros and cons of the argument are briefly as follows :—

Starches adhere to the teeth and are apt to remain there for some time, they are very freely taken at most meals daily in a very refined form, solid masses cannot ferment so rapidly as solutions, and the acid by-products of their fermentation would remain so localised that they would ultimately inhibit the bacterial growth which produced them.

Sugars in solution are far more readily fermentable, they are taken freely at most meals, and the quantity of sugar used per head per annum in the United Kingdom has been steadily increasing for the past two hundred years. Of late years the fermentable monosaccharide glucose has been increasingly used as a sweetening agent ; it is very readily fermentable. Caries often starts *around* the point of contact of one tooth with the next, as if the decalcifying agent had been held there in solution by capillary attraction ; also, caries is often found in pits and fissures which have been kept free of solid particles. Osborn's recent research,[1] however, indicates that it is the refined sugars chiefly that are so harmful, and that in fruits and sugar cane it is not so much the presence of the vegetable cellulose acting as a cleansing agent as protective substances present in the unrefined sugar which inhibit its decalcifying action.

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[1] *South Afric. Journ. Med. Sci.*, 1935, i, 57 ; *Journ. Dent. Res.*, 1937, xvi, 165.

The view that the acid is formed directly from the fermentation of food is not held by all observers. A. Lohmann [1] considers that the mucin is the agent which brings about the decalcification of the enamel, the mucin being set free from its combination with salts by the action of some acid. He states that: 'On account of the short stay of carbohydrates in the mouth, but a very slight transformation of the same can take place, whereby only maltose is formed. The production of lactic acid, however, presupposes the formation of grape-sugar, since cane-sugar undergoes no changes whatever in the saliva. Consequently, lactic acid cannot be formed in the mouth.'

Clinically, there is evidence to show that mucus is causally related to caries. Children who, in consequence of being 'mouth-breathers,' have developed a marginal gingivitis around the anterior teeth, show a tendency to caries on the approximal and labial aspects of those teeth, and this tendency exists even in mouths where every care is taken to cleanse the teeth by artificial means. Mucus secreted by the glands of the mucous membrane often has an acid reaction which is due, according to Kirk, to the presence of acid phosphates of lime and soda. It seems probable that this acid reaction is in some way related to the rapid caries of these cases, and the fact that, in mouth-breathers, the natural cleansing of the teeth is interfered with would appear to support this view.

It is, too, a clinical fact that caries is often found to be very extensive in patients who have viscid saliva, such patients as those in whom, on moving one's finger from salivary contact to a distance of two feet away, a thread of viscid mucous saliva maintains an unbroken connection.

The Chemical Action of Sugar.—Sugar has a strong affinity for calcium in its chemical capacity quite apart from the results of any bacterial action on it. Recently one of us (E. S.) [2] incubated, in some eight batches at blood heat, the suspended enamel-covered crowns of teeth in sugar solutions of known strength for three months, and separately, ground sections of teeth cut so as to show some cross sections of enamel prisms. After quite a short incubation period the sugar solutions, which had been carefully tested for neutrality and the presence of calcium beforehand and were checked by saline controls, showed the presence of calcium in them, which could only have been derived from the enamel. Ultimately, on trying to permeate these teeth with stains, but little penetration of stain from the outside was noted in the complete crowns, but in the sections it was found that the stain would now penetrate the prisms, but not the interprismatic substance (fig. 425), so that it appears that sugar can abstract calcium, probably by the formation of calcium sucrose, from the prisms, but not from the interprismatic substance, or anyway not so easily, the action appearing to be rather converse to the action of acid on enamel and indicating a chemical difference between these structures ; here also the action appeared to be superficial, as if the sections were ground too thin, the stain-penetrated position was lost. At first, as the whole crowns immersed in sugar solution became more transparent than normal and the tests showed that calcium had been

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[1] *Archiv für Zahnheilkunde*, June 1904.

[2] *Brit. Dent. Journ.*, 1938, lxiv, 245. (E. S.)

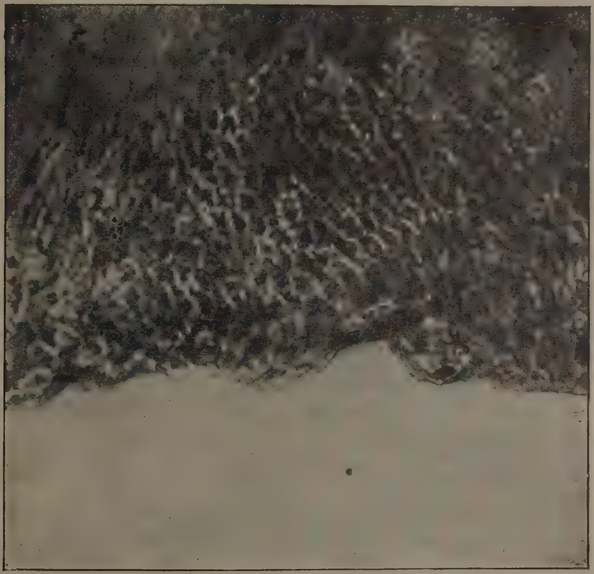

Fig. 425.—Young human enamel, ground after incubation of a thick section for three months in sugar solution. Shows absorption of stain by prisms but not by interprismatic substance. Sections transverse to long axis of prisms. × 235. (E. S.) (From *Brit. Dent. Journ.*)

taken from them, it seemed that this increased transparency might be explained by the approximation of the refractive index of the more highly calcified to that of the less highly calcified part, but it was not so, or not entirely, as sugar itself acts as a clearing medium, and on washing out the sugar the normal degree of opacity returned. An attempt was made to measure the difference in the degree of translucency, but the teeth were too dense to allow of this being done.

Whether this chemical action of sugar on enamel is of clinical importance is not easy to assess; but certainly three months' incubation did not make a vast change in the permeability of the enamel, though of course it may assist in the initiation of caries.

(D) EXPERIMENTAL REPRODUCTION OF CARIES.

The production of caries in teeth artificially was first successfully accomplished by Miller. Teeth which were perfectly sound, but of various degrees of density, were cut into pieces of different sizes and placed in a mixture of saliva, meat, and bread. This mixture, which was repeatedly renewed, was kept for three months at 98° F., at the end of which time there was definite caries of the teeth. With caries produced by artificial means there is no translucency, but translucency forms no essential part of the carious process. If the reaction of the solution in which the teeth are placed becomes alkaline, or if the teeth are exposed to the air or to the action of different articles of food, such as coffee, tea, tobacco, fruit, etc., shades of colour are produced similar to those found in the mouth.

M. Choquet has recorded [1] an interesting experiment in which he submitted normal dentine to the action of a pure culture of a micro-organism. From three carious teeth which had been filled he isolated five species of micro-organisms. From one of these he obtained a pure culture and applied it to a small cavity drilled in the incisor of a sheep, the culture being retained in position for nine months. The dentine at the end of this period was found to have undergone certain changes, namely, softening and alteration in colour. The appearances, however, were in no way typical of carious dentine, and it cannot be claimed that artificial caries was produced in this experiment.

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[1] International Medical Congress, Paris, 1900, reported in *Brit. Journ. Dent. Sci.*, November 15, 1900.

Reference has been made to the constant presence of two types of bacillus in carious dentine, the *Bacillus acidophilus odontolyticus* Types I and II. If teeth are immersed in broth inoculated with these bacilli, artificial caries is produced. The method adopted was as follows :—

'Non-carious teeth were placed in broth and 2 per cent. glucose broth in 3 in. by 1 in. tubes and sterilised ; various strains were then inoculated into the broth, *S. salivarius* in both ordinary broth and glucose broth, and *B. coli* in glucose broth being used as controls. Every eighth day the teeth were removed and placed into fresh uninoculated broth under sterile precautions, so as to approximate somewhat to the conditions in the mouth and prevent the teeth remaining continually in an acid medium.'

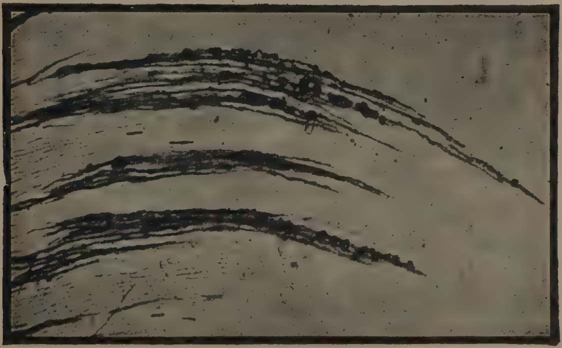

FIG. 426.—Artificial caries ; section showing spread of bacilli along the dentinal tubules, which are considerably dilated. × 40.
(From *Brit. Journ. of Experimental Pathology.*)

The longitudinal section of a tooth (fig. 426) which had been kept in glucose broth culture of Type I bacillus for fourteen weeks shows penetration of the dentinal tubes from the pulp cavity side.

The presence of liquefaction foci (fig. 427) is evidence of the power of the same organism to break up the collagen matrix of the dentine.

The action of milk upon dentine has been investigated by F. J. Bennett.[1] Sound teeth were broken into fragments and

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), iv, 3.

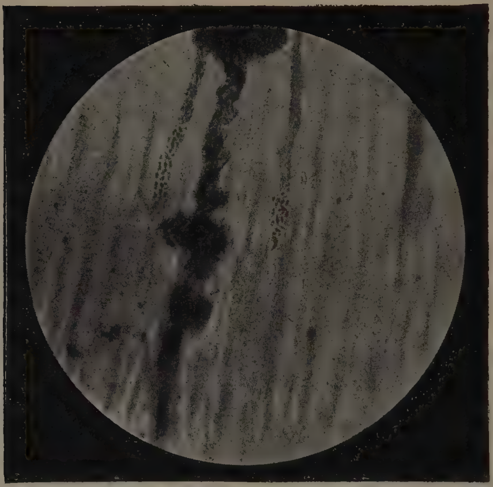

Fig. 427.—Artificial caries; individual bacilli in the dentinal tubules, also liquefaction focus. × 700.
(From *Brit. Journ. of Experimental Pathology.*)

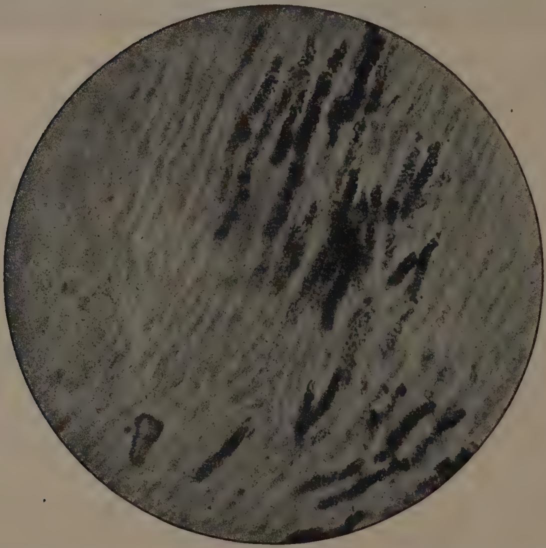

Fig. 428.—Portion of dentine decalcified by acid milk and stained to show micro-organisms in the tubes. (From *Proc. Roy. Soc. Med.*)

covered with 50 c.c. to 100 c.c. of cow's milk and kept in an incubator at 35° C. The milk was in some cases changed daily, in other cases every two or three days. In all cases the milk became curdled and showed an acid reaction to litmus in a few hours; this acidity was strongly marked in two to three days, after which an alkaline reaction set in with a characteristic putrescent smell. In no case was there an admixture of saliva or any other ingredient with the milk. The dentine became softened with patches of white opacity on the enamel. Sections of the dentine were cut on a microtome, and, on staining, the tubes were found to be occupied by organisms (fig. 428).

(E) FACTORS INFLUENCING THE INCIDENCE OF CARIES.

Under this heading various questions connected with the susceptibility and immunity of individuals to caries will be considered.

The Teeth.—Clinical and histological observations show that enamel varies considerably in density and permeability and in youth and age; it also varies in thickness and in the rapidity with which it breaks down when attacked by caries. These phenomena are probably intimately connected with the physical and chemical variations of enamel. Pickerill,[1] who has investigated the subject, divides teeth into three clinical groups:—

(i) *Native.*—Teeth of primitive races.

(ii) *Sclerotic.*—Teeth usually characterised by their 'hardness' and immunity to caries.

(iii) *Malacotic.*—Teeth characterised by their 'softness' and susceptibility to caries.

For the examination of certain markings on the enamel surface the following method of staining is adopted: The tooth to be examined is washed in water, dried, and the surface cleaned with xylol or ether and alcohol. Graphite is then applied on a soft pad and any excess removed by a piece of smooth rubber. The graphite is thus rubbed into all depressions, whilst it is removed from the elevations. By these means it can be shown that the surface of the enamel is traversed by a series of parallel grooves and ridges, to which Pickerill has given the name

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[1] 'The Prevention of Dental Caries and Oral Sepsis.'

'imbrication lines,' but they had been known for many years previously as 'Tomes's lines' (fig. 429). These lines are most marked at a point two-thirds of the distance between the neck and occlusal surface and are more defined in 'malacotic' than in 'sclerotic' teeth, and in 'sclerotic' more than in teeth of primitive races.

The imbrications are said to be intimately related to the striæ in the enamel, and caused by the outcrop of rows of prisms.

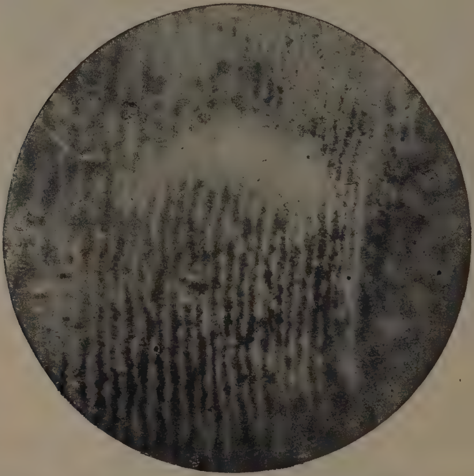

Fig. 429.—Imbrication lines on a malacotic tooth near the cervical region. (From 'Prevention of Dental Caries.' H. P. Pickerill.)

These striæ (of Retzius) coincide with a change of direction of a group of prisms, and seem to show up because the interprismatic substance is brought more in evidence by the prisms being shown in section transverse to their long axis where the striæ occur; at the same time it is generally believed that they do represent what was, at a given period of the laying down of enamel, the external surface of the amount then laid down, and so represent true incremental lines.

The view held by C. S. Tomes that the small unweighable amount of organic matter which he found present in enamel is negligible is untenable in the light of recent investigation. Lovatt Evans [1] estimated the organic content of enamel at 1 to 2 per cent., but admitted several possible sources of error in his estimation. One's investigations (with F. W. Bury) [1] into this question gave the organic content as 0·15 per cent. for deciduous and young and old permanent teeth, and this is quite sufficient to account for the histological evidence of organic matter in enamel to which reference will now be made.

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[1] *Trans. 17th Int. Med. Congress* (Sec. of Stomatology), 1913.

D. Caush [2] was the first to demonstrate that enamel is permeable and contains uncalcified material and that the quantity apparently varied in different teeth and in different individuals. The part which admits stain lies between the prisms and is termed by Caush interprismatic matter (figs. 430 and 431).

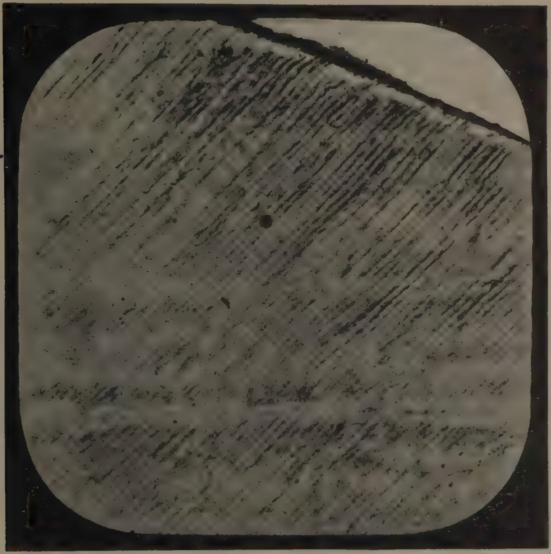

FIG. 430.—Permanent incisor. Interprismatic substance between the prisms, some terminating near the enamel margin where calcification is more perfect. × 200. (D. Caush.) (From *Brit. Dent. Journ.*)

Pickerill, too, has shown that the enamel is permeable to silver nitrate and that the permeability is greater in 'malacotic' than in 'sclerotic' teeth. An increase in the permeability probably lessens the resistance of the enamel and influences the progress of caries.

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[1] *Proc. Roy. Soc. B.*, 1928, cii, 419. E. S. and F. W. Bury.

[2] *Brit. Dent. Journ.*, xxvi, 97 and 901.

It is doubtful if the variations in the amount of uncalcified material can be regarded as entirely accounting for the remarkable difference in the hardness of enamel which is within the experience of every practitioner. The difference in the hardness of enamel is probably due to the quality rather than the quantity of the inorganic salt, but the subject is one which urgently needs accurate investigation.

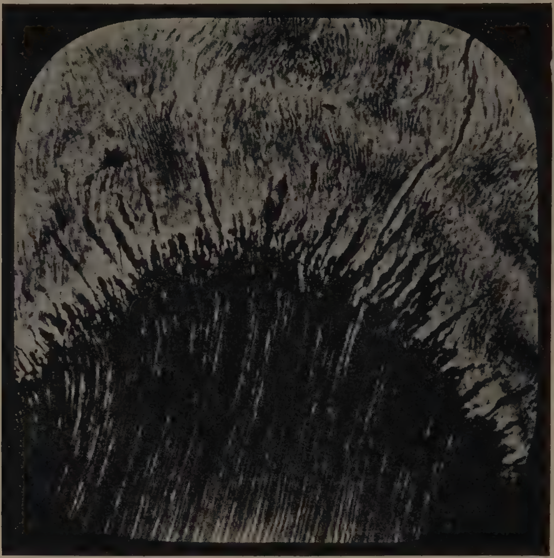

FIG. 431.—Spindles of Von Ebner and stained tubes in the enamel. × 100. (D. Caush.) (From *Brit. Dent. Journ.*)

In addition to exhibiting physical and probably chemical differences, enamel often shows macroscopical developmental defects in the form of pits and fissures, and such defects may in certain positions provide a lodging-place for food and so render the tooth more susceptible to caries.

Leon Williams[1] in his paper, 'The Pathology of Enamel,' states that defects in structure, such as pits, grooves, fissures, pigmentations, granular and amorphous enamel, are common in the lower animals. But the presence of such defects in the lower animals and the absence of caries, from which we know they are comparatively free, is no proof that these defects do not render the teeth more susceptible to caries, when the necessary pabulum exists for the production of lactic acid as in the case of human beings. In the majority of the lower animals the pabulum does not exist and hence no doubt their comparative freedom from caries. The horse may be taken as an example. In the maxillary teeth of the horse a defect is frequently found in the structure of the cemental tissue occupying the anterior and posterior 'lakes.' The defect takes the form of a canal which persists through the entire tooth (fig. 432), and when caries attacks the teeth of horses it invariably starts in the wall of this canal. L. A. Merillat[1] states that 'the entrance of food into these pits causes more than 95 per cent. of the decayed molars of the horse.' Horses kept on a diet of hay or grass are free from caries because the mouth bacteria have very slight action on cellulose material and

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[1] *Dental Cosmos*, March 1907.

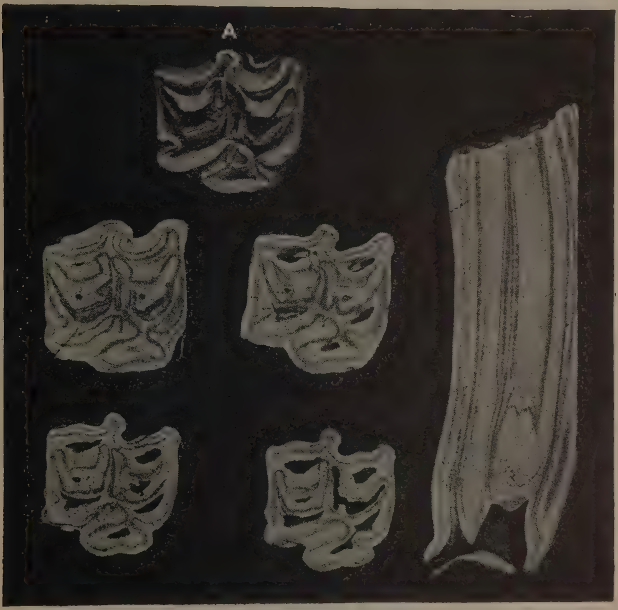

FIG. 432.—In the section (A) the opening of the canal is shown, with the surrounding tissue, in the early stage of caries; the other sections show that the canals exist throughout the tooth. (From *Trans. Odonto. Soc.*)

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[1] 'Animal Dentistry.'

lactic acid is not formed. When corn and other cereals are included in the diet a material is introduced from which lactic acid is formed, and the action of the lactic acid may produce caries which will start, as stated above, at the defective part of the tooth.

In fig. 433 is shown the section of a tooth which Miller immersed for nine and a half months in bouillon composed of water, 100 parts ; beef extract, 1·5 parts ; peptone, 1·5 ; sugar, 2 to 3 ; the solution being neutralised, sterilised, and infected with saliva. The tooth at the end of the period was found to be white and chalky, with the exception of a zone of 2 mm. (*a*), and on submitting the section to a 5 per cent. solution of HCl this zone was found to be the only part of the enamel covered with Nasmyth's membrane.

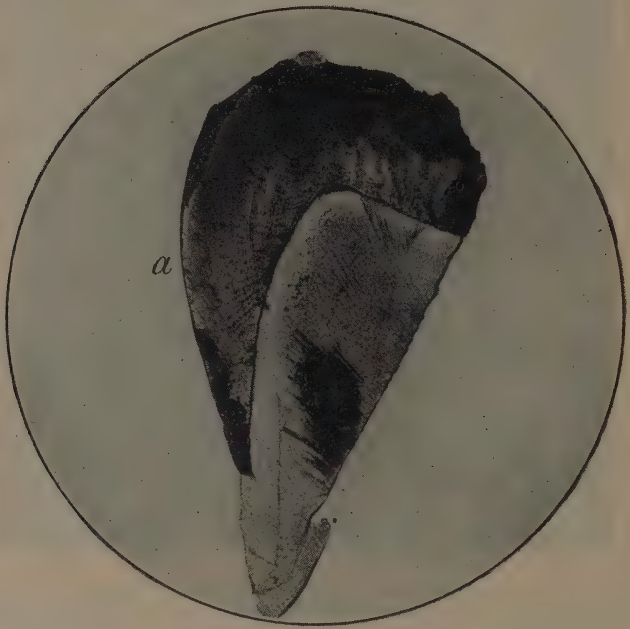

FIG. 433.—Section showing protective action of Nasmyth's membrane at (*a*). (From *Dental Cosmos.*)

On the approximal surfaces of teeth a patch of quite dark enamel will often be observed. In such cases *the carious process in the enamel has been arrested.* Miller has shown that these areas are peculiarly resistant. A tooth, a section of which is shown in fig. 434, was placed in a decalcifying solution for two months and then brushed repeatedly. The area of decay was more resistant to the action of the acid and brush than the normal enamel. To these areas of decay Nasmyth's membrane is said to be particularly tenacious. Miller also showed that 'the external intact smooth enamel surface offers a most stubborn resistance to the action of weak acids and far greater resistance than the internal surface.' (Fig. 435.)

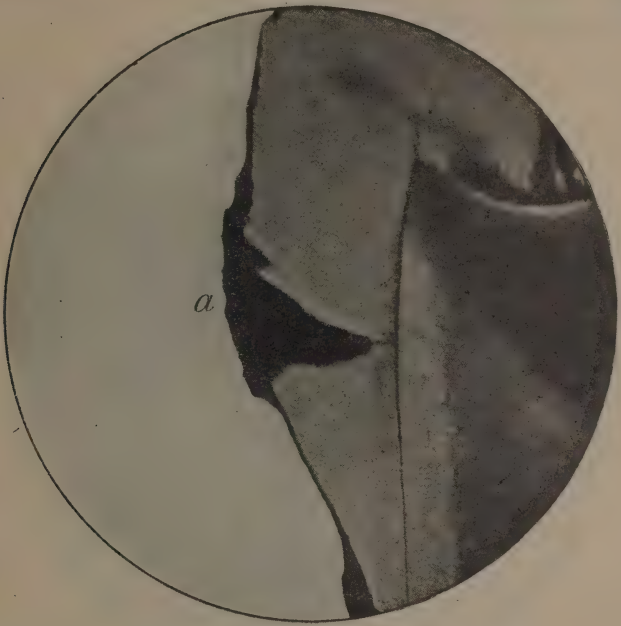

FIG. 434.—Decay mark (*a*) showing resistance to the action of acid and friction more than the sound enamel. (From *Dental Cosmos*.)

From the foregoing observations it seems possible that Nasmyth's membrane may protect the enamel to some extent.

G. V. Black[1] and C. S. Tomes[2] have published analyses of dentines and have stated that teeth, clinically known as soft, are not deficient in lime salts, although hitherto supposed to be deficient, and that the molars contain more inorganic material than the incisors. C. S. Tomes's figures are as follows :—

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[1] *Dental Cosmos*, 1895.

[2] *Brit. Dent. Journ.*, October 1895.

Incisors : 71·2 per cent. inorganic salts.
,, 28·8 per cent. organic matter and combined water.
Molars : 73 per cent. inorganic salts.
,, 27 per cent. organic matter and combined water.

Black's figures, reduced to percentage of dry dentine, give 71·7 for incisors and 72·3 for molars.

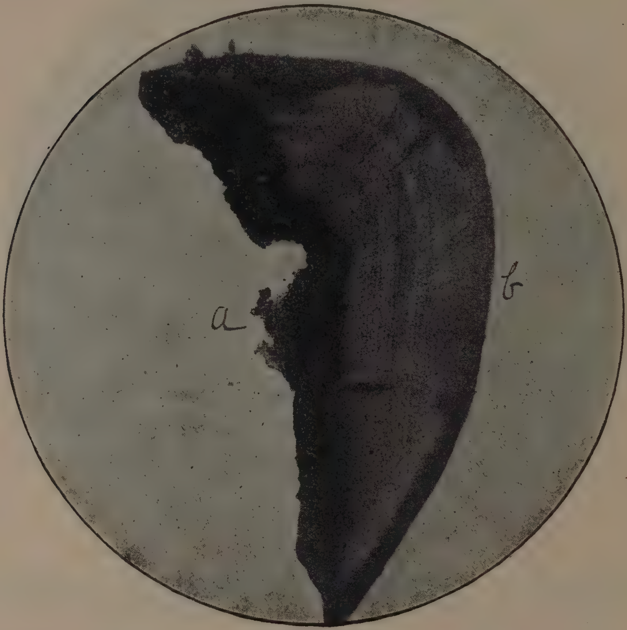

Fig. 435.—Unequal action of acid upon internal (*a*) and external (*b*) surfaces. (Miller.) (From *Dental Cosmos.*)

Two sets of teeth were analysed by Tomes to ascertain the percentage of salts. In one set the third molars were largely affected by caries and incipient decay existed between many of the teeth, while in the other set, although much worn by attrition, the teeth were quite free from caries. The analysis gave 71·4 per cent. of salts in both cases, which is below the general average. Tomes has also shown that both dentine and enamel contain water in chemical combination with the various lime salts.

Black[1] attached much importance to his own investigation, and considered that 'neither the density nor the percentage of lime salts, nor the strength, is in any degree a factor in predisposing the teeth to caries or in hindering its inception or progress.' The *important point, however, is not the percentage of lime salts in the teeth so much as the stability of the compound which these salts form with the organic matrix of the tooth.* The analyses of Black and Tomes are unfortunately in no respect qualitative and do not bring out the varying proportions of the different salts.

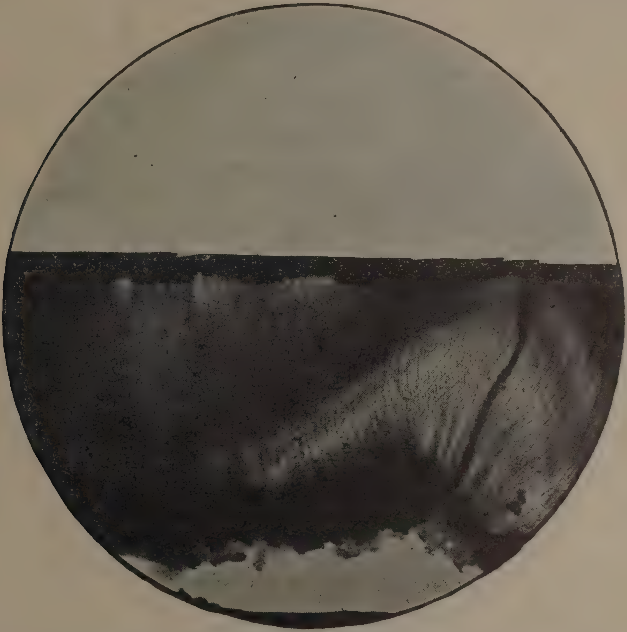

FIG. 436.—Showing the influence of transparent dentine in the process of decalcification. (Miller.) (From *Dental Cosmos.*)

Experiments on the resistance of dentine to acids were carried out by Miller.[2] The results he obtained force one to the conclusion that the resistance which dentine from the teeth of different animals offers to the actions of acids varies very greatly. Experiments with yellow teeth and weak bluish teeth showed 'that there is a difference in the rapidity with which dentine from different teeth is acted upon by weak acid under the same conditions, and that this difference will usually be found to be in favour of so-called hard dentine. The difference is not sufficiently great, however, to confer immunity upon the one tooth, while the other falls a prey to caries. It is only one of the factors, which again must not be completely lost sight of.'

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[1] *Dental Cosmos*, May 1895, 417.

[2] *Ibid.*, September 1903, 696.

The amount of reaction which takes place in teeth with living pulps doubtless influences the progress of the caries. The reaction of the tooth to the carious process manifests itself in the formation of translucent zones of hypercalcification in the enamel and in the dentine and the deposition of secondary dentines. Experiments show that the dentine of the 'translucent zone' and secondary dentine 'offer a greater resistance to advancing caries than normal dentine' (figs. 436 and 437).

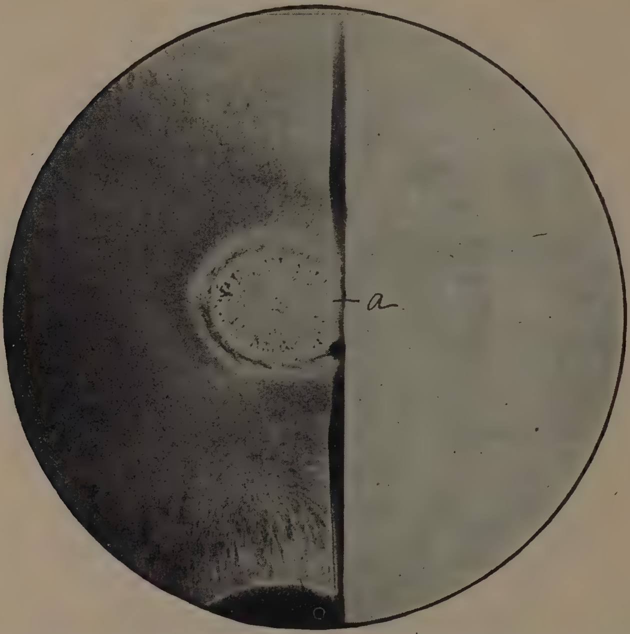

FIG. 437.—Evident superior resistance of secondary dentine to decalcification at (*a*). (Miller.) (From *Dental Cosmos.*)

The Influence of Lime Salts in Food.—Röse has made extended investigations as to the influence on the teeth of lime salts in food and water. His inquiries have been pursued in many countries, and he has arrived at the conclusion that in districts where the soil and drinking-water are rich in lime the teeth are sounder than in districts where the lime is deficient. The following figures show the results he obtained from Baden and Thuringia :—

(1) Country Places poor in lime.

	Percentage of children with caries.	Percentage of diseased teeth.
In Baden	98·7 ...	35·3
In Thuringia	92 ...	34·9

(2) Country Places rich in lime.

	Percentage of children with caries.	Percentage of diseased teeth.
In Baden	79 ...	16·1
In Thuringia	82·8 ...	16·7

Röse found a remarkable difference in the amount of caries in children at two villages, Guntersthal and Uffhausen, only about three kilometres apart. At the former, where the geological formation is gneiss and the water 1·7 to 2·8 degrees of hardness (German calculation), in 118 children 34·6 per cent. of the teeth were carious, while at Uffhausen, where a Jura-lime formation exists, and the water was 14 to 19 per cent. of hardness, in 162 children the percentage of carious teeth was only 20·7.

J. B. Cook [1] has investigated the subject in eighty urban districts in England and Wales. He finds that the harder the water the better the teeth, and, what is also of interest, that the harder the water the lower the infantile mortality. The relation of the hardness of the water to the incidence of caries must not necessarily be regarded as cause and effect but rather as a possible factor in the degree of relative immunity.

The effect of the calcareous content of drinking-water upon the incidence of caries in Switzerland has been investigated by P. Walter.[2] Two places were chosen, Stilli in Aargau, 328 metres above sea-level, with very hard water, and Engelberg, in Obwalden, 1,019 metres above sea-level, with very soft water. In Stilli the percentage of persons with carious teeth was 18 per cent., as against 36 per cent. in Engelberg.

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[1] *Lancet*, March 28, 1914.

[2] Quoted in *Brit. Dent. Journ.*, July 15, 1922, xliii, from the *Schweizerische Monatsschrift für Zahnheilkunde.*

The Type of Organisms present in the Mouth.—Investigations carried out by Pickerill and Champtaloup [1] show that in Maori children, who are comparatively immune to caries, the organisms present in the mouth are morphologically similar to those found in the mouths of individuals belonging to the more highly civilised communities; a fact which indicates that susceptibility and immunity to caries do not depend upon the bacteriological flora of the mouth.

The position of the teeth in relation to one another has an important bearing on their liability to caries. When they are in regular position the surfaces are kept free of food debris by the physiological action of the act of mastication, particularly when unrefined foods are eaten; under these circumstances, too, artificial means of cleaning are more efficient; but when they are placed irregularly, inaccessible crevices are formed. Again, the teeth, when normally placed, touch one another only at the crowns, and the space at the necks is occupied by gum which prevents the lodgment of food. Any agency, therefore, which operates to change these conditions—for example, causes a loosening or recession of the gum—acts as a predisposing cause of caries.

The Self-cleansing Action of Mastication.—If a series of mouths be examined about fifteen minutes after a meal it will be found that in some mouths the teeth will be entirely free from food, while in others there will be a considerable deposit around and between the teeth. It is obvious, therefore, that some mouths are more self-cleansing than others, and it is highly probable that the character of the individual in this respect affects the relative immunity or susceptibility of the teeth to caries. F. Breese [2] has recorded some interesting facts in connection with this question. In schools under the same dietetic regime and discipline with regard to artificial cleansing of the teeth, he found that the incidence of caries was in ratio with the self-cleansing action of mastication. The children were inspected immediately after meals and he found that (1) of 39 children (immune), in only 7 cases was food found and then only on the occlusal surfaces of the molars; (2) of 30 children (non-immune), 21 cases had food remaining on the teeth and in 7 of these there was marked accumulation.

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[1] *New Zealand Dental Journal*, March 1914.

[2] *Brit. Dent. Journ.*, June 16, 1913.

The Possibility of the Formation of Antibodies in the Teeth.—Von Beust[1] has shown that if the ends of the roots of freshly extracted teeth are immersed in a strong alcoholic solution of fuchsin the crowns will in time take on a reddish hue, which is due to the passage of the stain from the pulp cavity into the enamel. He therefore assumes that plasma ‘ can reach all parts, generally speaking, of the enamel and that this renders it possible, nay, even probable, that the immunity to caries observed in some teeth lies in the tooth itself and is the result of the formation of antibodies.’ He does not, however, exclude the saliva as an active factor making for immunity.

The Saliva.—A strong flow of saliva flushes the mouth and removes particles of food from the teeth. Miller affirms that he has repeatedly found that a cessation or a marked diminution in the secretion of saliva is followed by acute caries which attacks nearly all the teeth simultaneously and often extends upon the free surfaces. A viscid saliva may also conduce to caries by interfering with the self-cleansing of the mouth. It seems probable also that the susceptibility to caries may be influenced by the constitution of the saliva, for Miller,[2] as the result of many experiments, came to the conclusion that ‘ the saliva of those immune to caries develops in the presence of carbohydrates, in and out of the mouth, on an average a little less acid than that of highly susceptible persons. The difference is, however, not constant, and is not sufficiently marked to account for the marked difference of susceptibility.’

Röse[3] has analysed the saliva of 219 children whose average age was thirteen years, and he concludes that a distinct relation exists between dental caries and the alkalinity of the saliva. He finds that a high alkaline reaction constitutes the best means of checking the progress of caries. He has carried out a considerable number of experiments to determine the means by which slightly alkaline or acid saliva may be modified so as to render its reaction decidedly alkaline. He finds that a diet consisting of food-stuffs rich in calcium salts increases the alkalinity and quantity of the saliva, and he believes that such a diet favourably influences the quality of the teeth. In this connection it is interesting to note that Miller, in comparing, experimentally, equine and human saliva, found that in equine saliva no acid was produced during the first twenty-four hours, and that the total amount at the end of nine days was 50 per cent. less than in human saliva. Equine saliva is strongly alkaline and therefore more readily neutralises any acid formed. In Chapter XIX reference is also made to the part played by saliva in relation to caries of the teeth.

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[1] *Dental Cosmos.*

[2] *Ibid.*, 1903, 689.

[3] *Deutsche Monatsschrift für Zahnheilkunde*, December 1905.

The Relation of Sulpho-cyanide of Potassium to Caries.—Opinions differ as to the relation of sulpho-cyanide of potassium to caries. This salt is a normal constituent of saliva, forming about 0·1 of the total solids. Gies[1] has made a thorough investigation into the subject and finds that 'there is nothing about the known quantities of sulpho-cyanide to indicate that it is able, *in the proportions of its normal occurrence in saliva*, to affect the secretory tendencies of the salivary or buccal glands, to modify the oral membranes, to influence the teeth from any standpoint, or to stimulate or retard or alter the activities of the oral micro-organisms.'

Blood Groups.—Professor V. Suk,[2] after examination of some 700 young men aged from 18 to 22 years, mostly students and soldiers, finds that the incidence of caries varies in the different blood groups. Thus, those whose blood belonged to Group O showed the highest percentage of teeth without caries. In the O group 26·8 per cent. had good teeth, in the A group 18·9 per cent., in the B group 16·2 per cent., and in the AB group 21·8 per cent. He thinks the differences are significant, and states that perfect teeth and the O group are both more common among primitive people than among whites and that the white races are losing this quality as civilisation advances.

Vaccination.—So profound a thinker as Herbert Spencer hazarded the suggestion that vaccination might increase the susceptibility of the teeth to caries. This view is shared by a few dental practitioners. D. V. Peacock[3] considers that vaccination is a prime factor in the destruction of children's teeth, his argument being that it leads to imperfect development of the tooth structure. Reliable statistics in support of this view are not, however, forthcoming.

Sex.—The work of Röse, Broughton Head, one's own[4] and that of others all tends to show that the incidence of dental caries is slightly higher in female children than in males of the same age and living under the same conditions. The reason for this is that teeth tend to erupt rather earlier in females, and have therefore been exposed longer to whatever causative factors of dental caries have been present.

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[1] *Dental Cosmos*, February 1914.

[2] Publications of the Faculty of Science of the Masaryk University, No. 125.

[3] *Dominion Dent. Journ.*, December 1901.

[4] *Trans. of School Dent. Soc.*, 1914, vii, 6. (E. S.)

Pregnancy.—It is a current belief that there is increased susceptibility to caries during pregnancy, and it has been assumed that this is due to the absorption of the mineral matter from the teeth to form fœtal bone, and although this is a period when calcium starvation may occur, there is no evidence that mineral matter is ever so absorbed from the teeth. From a physiological point of view there would appear to be no foundation for this belief. S. Biro [1] has examined the teeth of 200 mothers and the results he obtained tend to show that pregnancy does not cause any increase in caries of the teeth. During pregnancy certain women often seem more liable to caries, but an explanation may be found in some change of the oral secretion whereby a more favourable soil is produced for the development of micro-organisms; or it may be that the altered nervous state which often exists causes dental lesions to give rise to signs and symptoms when otherwise they would not do so. It has been thought that the vomiting of pregnancy by bringing acid gastric juice into contact with the teeth may initiate their decalcification, but many women have no pregnancy vomiting, and among those who do it exists in the majority during the second and third months of pregnancy only, and often consists merely of early morning retching; moreover, gastric juice only contains 0·2 per cent. of hydrochloric acid and its effects would be very transient, probably much more so than the drinking of a glass of lemonade. This alleged predisposition of the pregnant to caries is by no means a matter of general experience, and in practice one meets with many instances of women with large families who have excellent teeth and who develop no increased susceptibility to caries during the pregnant period.

H. Klein [2] in an examination of the extensive human and comparative literature of this subject comes to the conclusion that: 'The data on humans indicate that the incidence of tooth decay increases with advance in age, and is not related to the experience or the number of pregnancies.'

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[1] *Vierteljahr. f. Zahnheilkd.*, 1898, xiv, 371.

[2] *Dental Cosmos*, 1935, 77, 864.

The Influence of Feeding in Infancy.—Kingston Barton,[1] as a result of twenty years of observation, finds that breast-fed children have the best teeth ; those fed on cows', asses' or goats' milk come next, and when starch or any patent food is added to or given in place of cows' milk, the teeth, both deciduous and permanent, almost always suffer severely. The figures published in the report of the Salop County Council show that the incidence of caries in breast-fed children is only slightly less than in the bottle-fed. The figures are as follows :—

	Breast-fed Children.		Bottle-fed Children.	
Age.	Number of children examined.	Number of carious teeth per child.	Number of children examined.	Number of carious teeth per child.
5 to 6	5,707	6·7	3,375	7·1
12 ,, 13	3,571	4·7	1,900	5·0

Adenoids.—Statistics show that caries is more prevalent in mouth-breathers than in nose-breathers, and this is no doubt due to the facts that in the mouth-breathers (1) food debris is more liable to cling around the teeth, and (2) the number of micro-organisms is increased. The following figures published by H. R. Burpett[2] are of interest :—

	Children with adenoids.			All children.		
	Infants Per cent.	Intermediates Per cent.	Leavers Per cent.	Infants Per cent.	Intermediates Per cent.	Leavers Per cent.
Teeth excellent	7	10	12	13·2	12·6	18·5
Four or more teeth carious	33·5	28	12	26·4	21·4	10·4

Poverty.—In discussing this aspect of caries, comparisons will be drawn between the various classes of children who attend State schools. Statistics show that amongst such children the poorest had the best teeth. The following figures are taken from an inspection of children in Staffordshire[3] :—

	Age 5–6		Age 8–9		Age 12–14	
Sound dentures among—	Number examined.	Per cent.	Number examined.	Per cent.	Number examined.	Per cent.
(*a*) All children coming from houses, one to three rooms	478	23·2	379	12·6	159	22·6
(*b*) All the other children attending the same schools as the children in (*a*)	2,434	20·2	2,445	7·5	1,025	18·2
(*c*) All the children of all the schools of the county	6,485	18·3	6,198	8·4	3,421	18·9

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[1] *Medical Press and Circular*, 1899, ii.

[2] Quoted in *Dental Record*, November 1916, 603.

[3] Annual Report of the School Medical Officer, Staffordshire County Council, year 1911.

The general high percentage of sound dentures requires a word of explanation. In the inspection no probe or mirror was used; hence many carious cavities must undoubtedly have been overlooked. The figures, however, are comparative and distinctly show that the poorest children had the best teeth. The poorest children had the most simple food, while lack of means (in 1911) prevented the purchase of cakes and sweets, and the better preservation of their teeth was probably due, therefore, to the comparative absence of the highly fermentable and refined carbohydrates from their diet.

Food Deficiencies.—The work of M. Mellanby on dogs has shown that food deficiencies may affect the structure of the dentine profoundly in that there may be excessive formation of interglobular spaces; the enamel seems to be affected chiefly as to the amount (thickness) of it which is formed. On the other hand, administration of the calcifying vitamin D while the teeth were developing was said to cause thick enamel and dentine without interglobular spaces to be formed; these structures may even have been hypercalcified. It is often denied that such teeth prove more resistant to caries than others, and as vitamin D is only one among many necessities in the formation of the perfect tooth it is not logical to suppose that immunity to caries should be attained simply by its administration, and indeed we know it is not.

Indeed there is the evidence of Friel and Middleton Shaw[1] and others, that people who live with large areas of their skin exposed to direct sunlight, even when the rays have great intensity of ultra-violet light (which produces vitamin D by its action on exposed skin or the ergosterol contained in it) have a very similar incidence of dental caries to those who are clothed.

Goll[2] studied the result of the administration of cod liver oil to young children with carious teeth and found that it had no effect at all on the prevention of further caries.

Taylor and Marshall Day,[3] and Wilson[4] give an account of children in the Kangra Valley of Northern India in the Himalayan foothills where there are: ‘a high incidence of rickets and osteomalacia, and definite vitamin D, calcium, and phosphorus deficiencies in the diet of most of the population there.' Wilson writes: 'In an examination which was carried out there it was found that twenty-four out of a total of forty-five children showed signs of rickets, late rickets or osteomalacia. The same children had almost perfect teeth, with a very low incidence of caries and little hypoplasia. The teeth of these children were, in fact, no worse than those of 938 children in the Central Provinces living on a similar diet and among whom no case of rickets was discovered. These dental findings have been confirmed by Dr. Marshall Day, who examined fifty children in the Kangra valley, all of whom had severe rickets, but all of whom he found to have excellent teeth.'

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[1] *Brit. Dent. Journ.*, 1931, lii, i, 309.

[2] H. Goll, *Wiener Klinische Wochenschrift*, 1939, 52, 35.

[3] G. F. Taylor and C. D. Marshall Day, *Brit. Med. Journ.*, 1939, i, 919.

[4] D. C. Wilson, *Journ. of Physiology*, 1939, xcvi, 6.

It is by no means rare to find an absence of caries when many hypoplasic teeth are present, and the enamel of such teeth is notably thin or deficient in quantity; but one's own work on hypoplasic teeth shows that even when young the enamel of these teeth may be quite impermeable to diffused stains, and so, apparently, quite highly calcified.

There is no evidence that a high degree of calcification is in itself any protection against dental caries; indeed it is very probable that the quality and stability of the calcium compounds are much more important than their being present in a high percentage, and the shape of the teeth, and the physical and chemical characters of the foodstuffs more important still.

Vitality of Enamel.—One's recent work,[1] which led to the conclusion that human enamel was within the pale of nutrition and reacted to injury, and became more highly calcified as age advanced after the eruption of a tooth, leads one to speculate on how this knowledge may affect the incidence or progress of dental caries, because if enamel can react to traumatic injury it would seem reasonable to expect it to react to chemical injury. The work of J. H. Mummery [2] in describing translucent zones of hypercalcification in enamel, and one's own, tells one that it does so react; as to whether such reaction can be made effective is doubtful, as one has no knowledge of the rate at which such hypercalcification takes place in erupted teeth.

A. MacGregor's [3] recent work, on tracing the lymphatic system of the jaws and teeth by the absorption of basic lead

[1] *Proc. Roy. Soc.*, 1930, B., cvi, 376. (E. S.)

[2] *Brit. Dent. Journ.*, 1926, xlvii, 473.

[3] *Proc. Roy. Soc. Med.*, 1936, xxix, 1237.

acetate, is confirmatory of enamel being within the pale of nutrition, as in a monkey he found that several dentinal tubules entering the enamel contained precipitated lead sulphide.

One can say that teeth are most prone to become carious during the years immediately following their eruption, at that period when their enamel is most freely permeable ; it may be that their relative subsequent immunity is partly[1] dependent on the higher degree of calcification which later on takes place in the enamel, particularly in the more external zone. If that is so, then, if such high degree of calcification can be expedited, its production should soon show in a lowered incidence of caries ; but if perfection is to be attained in the bulk, quality, and degree of calcification of enamel it must be produced while the tooth is developing, as one doubts if the teeth can be influenced to any great extent after eruption, and quality is almost certainly of more importance than degree of calcification.

A tooth is in this matter comparable to the child as a whole ; if the child is well and correctly fed and exercised its development is robust, but if its feeding and upbringing are faulty many of the effects so produced, such as small size, poor resistance, etc., will be present permanently, no matter what the perfection of feeding and environment may be after growth is complete.

The effects of the shortage of Carbohydrate Foods during the War.—During the war, shortage of carbohydrate foods occurred so that jams, sweets, biscuits, cakes, sugar, and white bread were with difficulty obtainable ; the effects of this were noticeable by those who returned to treat the teeth of groups of children in the same institutions at which they had worked before the war. One's own experience was that there was a marked decrease in the incidence of dental caries ; this had been carefully tabulated before the war in some 2,000 children, and in 1919, at the same institution, children of the same age showed an increase of 17 non-carious complete dentures per cent. The decrease in the incidence of caries was commented on by others about the same time.

Disordered Endocrine Function.—The opinion is held by F. W. Broderick, that acidosis brought about by disordered endocrine function or faulty dietary is an important etiological factor in the incidence of dental caries ; the evidence on this point, however, is not at present conclusive, so one must regard this condition as a predisposing rather than a causative factor, but it is a matter that needs further research.

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[1] There are, of course, other factors, such as the wearing away of pits, crevices, and fissures by attrition, which do away with stagnation areas.

He considers that the conditions requisite to produce dental caries and parodontal disease are opposed to each other, so that both cannot actively progress together though they may do so alternately.

Occupations and Food Idiosyncrasies.—Certain occupations are often found associated with caries in many teeth: chiefly these are workers engaged in bakeries, flour mills, and confectioners' shops, where the association with refined carbohydrates will be noted; one has also seen rampant caries in a professional chocolate taster; it also occurs in workers in chemical factories engaged in the manufacture of acids, such as tartaric and sulphuric, and possibly others. Occasionally, such food idiosyncrasy as the frequent sucking of lemons or other acid fruits predisposes to dental caries by initiating a preparatory decalcification and so facilitating the entry of acid-producing or proteolytic organisms.

Structural Defects.—Structural defects such as in fig. 409 also predispose to dental caries by providing stagnation areas which are uncleansable by either physiological or artificial means.

Absence of Caries-producing Organisms.—It is claimed by R. W. Bunting that a diet composed largely of unrefined foods and but little free sugar can induce freedom from *Bacillus acidophilus odontolyticus* and other caries-producing organisms in the mouth and so prevent dental caries, and he produces some evidence, in so far as his work has proceeded, that this is so. It may be noted that such diet not only produces no pabulum for the micro-organismal growth, but by its physical character would prevent such pabulum from lodging even if it were present. For the details of such prophylaxis of caries as may be brought about by diet, see Chapter XI.

Note.—Robust health and good teeth are usually coincident. Opinions have been expressed that one is dependent on the other, but have differed as to which is dependent on the other; it is probable that neither is so, but that both are dependent on the same common factors.

(F) ETIOLOGY.

In the preceding pages it has been shown that caries is a process which is started by solution of the inorganic matter of the teeth and continued by the peptonising action of bacteria on the remaining organic tissue, and that the acid or acids which cause the damage are formed from the carbohydrate food undergoing fermentation in contact with the tooth substance.

We have seen that caries existed as far back as the time of Rhodesian man. In the early stages of civilisation a definite increase in the amount of caries can be traced, and with the spread of civilising influences the disease has made more and more rapid progress until at the present day it is so prevalent that few are to be found with teeth entirely free from caries.

Caries in primitive races was probably due to circumstances and conditions unlike those which led to the extensive destruction of teeth at the present day. In primitive races the disease was of a more chronic type and usually followed injury of the dentine or cement—in other words, it did not arise, as in the present day, as a direct attack on the enamel.

B. Nicholls [1] has drawn attention to the close relationship of attrition of the teeth to caries in the aboriginal Australian race, and he says that in his investigation ‘ approximal caries was often associated with approximal and occlusal cavities in adjacent teeth, in which pulps had been exposed by excessive attrition.’ He also remarks that dental caries in aborigines is mainly a disease of middle and old age, and one of its chief predisposing factors is ‘ natural ’ destructive attrition.

Mummery in his investigations paid particular attention to this question of destructive attrition, and showed that attrition is not an index of hard food but rather of the degree of hard matter, such as sand, incorporated with the food and not eliminated in its preparation.

When there is destruction of the teeth by attrition the enamel is liable to flake off and so lead to a catchment area. The Eskimo, Maori, and North American Indians (Coast) skulls examined by Mummery showed a considerable degree of destructive attrition, but caries of the teeth was rare. The liability to caries in races showing extreme attrition of the teeth would seem to depend upon the type of food which is collected in the catchment area.

In referring to caries in animals, attention was directed to the fact that, when seen in skulls of animals from the wild state, the caries seemed to follow either injury of the cementum at the cervical margin or exposure of the dentine by fracture of the teeth; whereas in the animals kept in captivity the enamel was generally the part attacked. It seems possible that the destruction of the cementum and dentine seen in caries in some primitive races and in animals in the wild state may be of the nature of an autolysis, or may be due to proteolytic action, and not to the formation of lactic acid from carbohydrates.

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[1] *Trans. Sixth Inter. Dent. Congress*, 79.

The phenomenal rapidity with which caries has increased during the last few decades has puzzled those who have studied the subject. Considerable light has, however, in recent years been thrown on the problem by those who contend that a solution is to be found in the altered character of the food-stuffs. In a series of valuable papers, Sim Wallace has drawn attention to the influence which the refinement of food has had on the process of mastication, and he has clearly pointed out the intimate relationship which this change bears to the incidence of caries. To appreciate the standpoint taken up by this writer, reference must be made to the process of mastication of food of a soft character, and of food containing coarse or fibrous material. In the former case 'The food is simply taken into the mouth, receives a general squash between the teeth, or between the dorsum of the tongue and the hard palate, and is then swallowed. This method of mastication—if mastication it can be called—is, as a rule, adopted for custards, fine meal porridge, soft puddings, and soft non-fibrous foods generally. When there is a certain amount of coarse or fibrous matter in the food-stuff, the process is essentially different, and mastication is performed in a more thorough manner. The food is crushed and torn between the teeth and heaped on to the masticating surfaces by the muscular contractions of the tongue, cheeks, lips, and motions of the lower jaw. During comminution between the teeth, the juices of the food-stuffs, the saliva which becomes incorporated, and the suspended non-fibrous part are pressed out from the fibres and gradually collect during the process on the middle of the dorsum of the tongue, which is gradually hollowed out for the reception of such food, and this portion of the food is then swallowed. The fibrous part of the food, however, is subjected again and again to the crushing and disintegration between the teeth. If any of the fibrous part passes towards the back of the dorsum of the tongue, it is arrested by the pressure of the anterior part of the tongue against the rugæ of the palate, and while the fluid and finely comminuted part gets sucked or pressed back into the hollow formed at the back of the dorsum of the tongue, the coarse and fibrous parts are thrown between the teeth and subjected again to the crushing, squeezing, and comminution. The rough surface of the tip and dorsum of the tongue and the smooth-ridged palate are especially well adapted for separating the food which is prepared for swallowing from that which requires further mastication.'

'If this process is carefully observed, it will be found that the food is not, as a rule, formed into a bolus and then swallowed, but that the bolus is continually slipping away to the dorsum of the tongue, to be swallowed only when there is almost or quite no bolus left.' (Sim Wallace.)

The recognition of the real nature of the process of mastication is, in the authors' opinion, of great importance, for the effect of chewing food of a more or less fibrous nature tends to dislodge the fine particles of food-stuffs which otherwise are apt to remain between and on the teeth.

If we examine the food-stuffs as prepared for the table at the present day, we find that every care has been taken to eliminate the fibrous element, and that they are usually presented in such a soft condition as to require very little mastication; indeed, it is extremely difficult, even with the best intentions, to masticate such food. The process of mastication, therefore, remains in abeyance, and the result is that, owing to the pappy character of the food and the absence of the cleansing operation of mastication, the food tends to cling around the teeth. Sim Wallace contends that herein lies the true cause of the prevalence of caries. He says: 'The cause of the prevalence of dental caries is that the natural food-stuffs are to a large extent ridded of their accompanying fibrous parts and prepared and consumed in a manner which renders them liable to lodge and undergo acid fermentation in the mouth; while from the same cause and the induced condition the micro-organisms of the mouth lodge, multiply, and augment the rapidity and intensity of the acid fermentation.'

In opposition to these views it is argued that there are numerous examples of races living upon soft food and yet not unduly liable to caries. The Kaffirs of South Africa are given as an instance. But the porridge of the Kaffirs, which is their staple food, is prepared by bruising and crushing mealies, no attempt being made to eliminate the coarse and fibrous parts, and experience shows that such food *does* require a considerable amount of mastication.[1]

S. Colyer, who has lived for several years among the races of Central Africa, states that the diet of many of these people is mainly 'grain,' and that it is prepared for eating in a 'stodgy, elastic and rather sticky mass,' which requires but little mastication. The natives do not clean their teeth after meals, and their diet is practically free from 'acid and saltish savouries.' The food, however, is practically free from easily fermentable carbohydrates, in the form of the various sugars. These races have very little caries, and it would therefore seem that caries cannot be solely attributed to soft, sticky, starchy foods.

During the last sixty years the increased and varied use of carbohydrates has probably had an important influence in accelerating the incidence of caries. *Carbohydrates as used at the present day are more easily soluble and fermentable than heretofore*, and have undergone an extraordinary amount of variation. Flour is a typical example. In flour the change has been brought about by the altered method of milling. Milling with the old stone mills excluded no part of the grain except the greater portion of the husk, whereas with roller milling the germ is removed and practically all the bran, the flour consisting almost entirely of starch and gluten. Flour so produced was introduced into this country as 'Pure white Hungarian flour' about 1875, and the bread made from it appealed to the public because being white it was thought to be purer and cleaner; it has remained largely in favour ever since. With regard to the question of caries, bread made from stone-milled flour requires more mastication and insalivation than that made from the roller-milled, and what is probably more important still is that the much finer subdivision of the starch granules in the roller-milled flour offers an increased surface for the action of fermentation.

The increased consumption of biscuits is probably in some measure responsible for the prevalence of caries. In biscuits the carbohydrate is in a peculiarly sticky form, and all the pabulum necessary for the rapid production of lactic acid is present in the mouth. The rapid and extensive caries seen in young children can often be traced to biscuits given just before going to rest.

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[1] References to this question will be found in the *Lancet*, 1900, September 8, 770; November 3, 1307; *Brit. Med. Journ.*, 1905, March 18, 629; April 1, 749.

There is abundant clinical evidence to show that the use of sugar is closely connected with the amount of caries. The use of sugar as a food-stuff has increased enormously during recent years, and this food, it must be remembered, is readily soluble.

The harmful effect of sugar on the teeth is referred to by Berdmore [1] in these words : 'I am credibly informed that in the Low Countries where sugar, tea and coffee, and sweetmeats are used in excess, the people even at an early age are remarkable for the badness of their teeth. It is therefore advisable to eat of them but seldom, and always to wash the teeth after them.' Hentzner, the German traveller, noticed that the teeth of Queen Elizabeth 'were black, a defect the English seem subject to from their too great use of sugar.'

Sugar reached England about the beginning of the twelfth century, but it was only a luxury indulged in by the well-to-do classes. At the beginning of the eighteenth century the average amount per head imported was 3 lb. ; at the beginning of the nineteenth century, 22 lb. ; in 1905 the amount had risen to 74·45 lb., in 1911 to 92·05 lb., and in 1928–29 to 101·3 lb. In 1911, 6·31 lb. per head of glucose was consumed in addition to the imported sugar. These figures show that the use of sugar as a food-stuff has greatly increased during recent years. The facts that about eighty to ninety years ago sugar cost about one shilling per pound and that wages were then considerably lower than at present have obviously a considerable bearing on this increased consumption.

The recent increased use of the monosaccharide glucose and its frequent prescription by physicians has not helped to decrease the incidence of caries, as it is directly fermentable in the mouth ; dietetic experts now say that cane sugar has precisely the same therapeutic value, as all sugars have to be converted into glucose before they can be absorbed, and the conversion of cane sugar into glucose requires the minimum of digestive effort.

The result of S. Colyer's [2] observations of certain African tribes supports the view that fermentable sugars are an important factor in the production of dental caries :—

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[1] 'A treatise on the Disorders and Deformities of the Teeth and Gums,' Thomas Berdmore. 1768 (First Edition).

[2] *Dental Record*, xxxvi, No. 1 ; xxxvii, No. 9 ; xxxix, No. 11.

(1) The Barotze, a people living on either side of the Upper Zambesi, have as their chief food cassava and mealies. Cassava is a shrub with large tuberous roots, which, after soaking and drying, is pounded into a fine white powder, possessing a large, but very finely divided, fibrous constituent. It is prepared for eating by sprinkling the powder with water which has been just brought to the boil and then stirring with a stick, the resultant mixture being an elastic and rather sticky mass. Mealies are also prepared as a thick glutinous mass. The food requires very little mastication and tends to cling about the teeth, which are not cleaned after meals. Now, amongst the Barotze caries is uncommon, while marginal gingivitis and parodontal disease are extremely common. The caries which does occur progresses slowly, and seldom if ever starts on the approximal surfaces of the teeth.

(2) Amongst the natives living near or upon the Lakes Mweru and Bangweolo, whose staple food is red millet, parodontal disease is very common, but caries is even less common than in the Barotze, only one tooth in 270 being carious. The comparative freedom from caries in these two groups, S. Colyer remarks, cannot be due to the self-cleansing action of the foods, for they are soft and sticky; nor can the immunity from caries be attributed to the absence of carbohydrates, for the foods eaten contain a high percentage of this element: cassava about 20 per cent., maize and millet about 70 per cent. These food-stuffs, however, do lack the presence of easily fermentable carbohydrates in the form of the various sugars.

(3) In the Transkeian territories the staple foods are mealies and Kaffir corn, but in addition *a considerable quantity of sugar is eaten*; the sugar is mixed with the porridge, is sometimes taken raw, and two, or at times three, teaspoonfuls are added to each cup of tea or coffee consumed. Mothers give sugar to their children by day and sometimes at night to pacify them. Amongst these natives, caries of the teeth is common, the interstitial surfaces being frequently attacked. Natives are seen with dentures completely ruined by caries. Caries of the incisors is common, and the young are as frequently affected as their elders. Parodontal disease, on the other hand, is comparatively rare.

In a paper by L. Ottofy on 'The Teeth of the Igorots,'[1] the relation of sugars to caries is well brought out. In the Igorots of the Philippines, a semi-barbaric race, he found only 2·05 per cent. of carious teeth ; while in the Filipinos, the more civilised inhabitants of the islands, the amount of caries was 20·9 per cent. The diet of the Igorots consists of food requiring considerable mastication ; bread is unknown and sugar is distasteful to the children. The Filipinos, on the other hand, indulge freely in the practice of chewing sugar, which is sold in small pieces all over the islands, and Ottofy is convinced that the prevalence of caries in the Filipinos is due to sugar. In practice it is found that children who eat sweets in large quantities will return from school each term with a plentiful supply of fresh and generally rapid caries, but when the eating of sweets is stopped the amount of fresh caries rapidly declines.

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[1] *Dental Cosmos*, July 1908.

In the county of Shropshire an inquiry was carried out to ascertain the relation of ‘ sweets ’ to caries, the term ‘ sweets ’ being used in the popular sense and not including sugary foods taken at meal times. The classification adopted was :—

Class 1. Large quantities of sweets eaten—almost every day.
Class 2. Considerable quantities eaten—several times a week.
Class 3. Few sweets eaten—about once a week.
Class 4. No sweets at all eaten.

In all 5,856 children were examined, and it was found that caries predominated where sweets were eaten in large quantities, and that as the consumption of sweets diminished the number of carious teeth decreased and the number of sound dentures increased.

Under the nomenclature of ‘ comforter caries ’ E. H. Harries [1] drew attention to a rapid destruction of the maxillary incisors in infants, the teeth becoming affected soon after their eruption. In some of the cases thickening of the upper lip was noted. All cases were associated with persistent use of the comforter. In cases which have come under one’s observation the comforter has usually been coated with a sweet substance, and it would seem therefore that in these cases the caries is not due to the action of the comforter itself but to the fermentation of the sugary substances used to cover the comforter.

Recapitulation.—The process of caries may be concisely described as follows : The form and relative positions of the human teeth favour the lodgment of food-stuffs in solid form or in solution upon or between the teeth. Of the groups of food-stuffs, the carbohydrates are concerned in bringing about the process of caries. Moreover, they constitute the only food which civilised man takes in forms essentially different from those taken by wild animals among whom caries is relatively rare.

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[1] *Lancet*, 1911, November 11, 1327.

Of the three groups of carbohydrates—cellulose, starch, and sugar—it is doubtful whether the first (cellulose) undergoes any change in the mouth which gives rise to products injurious to the teeth; whereas starch is first converted into dextrose by the action of the ptyalin of the saliva, and the dextrose, through the agency of micro-organisms into lactic acid, and possibly other organic acids in minute quantities. The sugars, when directly fermentable, are by the same process converted into acids, whereas those not directly fermentable, such as cane-sugar, take up a molecule of water under the action of invertin (a ferment produced by many bacteria), and are then split up into dextrose and levulose, both of which are directly fermentable, and produce lactic acid in the presence of many mouth bacteria. It is known that the first stage of dental caries is one of decalcification, and that carbohydrates are the only food which can undergo fermentation and produce acid in the mouth; it is therefore only logical to conclude that acid formed from carbohydrate fermentation in contact with the teeth starts the carious process.

These acids attack the teeth at the point where they are generated, that is, wherever the foods in solid or fluid form have lodged on or between the teeth, and lead to decalcification, first of the enamel (or cement), and later of the dentine. The decalcification of the enamel results in its complete destruction, as the organic substance in the enamel is insufficient to hold together after decalcification, whereas decalcification of the dentine does not imply the complete destruction of the organic basis, as this remains in the form of the so-called tooth cartilage. As this organic basis is of a nitrogenous nature it undergoes decomposition in very much the same way as all other nitrogenous substances exposed to the action of bacteria in the presence of moisture and a suitable temperature. The agent which brings about this decomposition is a ferment produced by many of the bacteria of the mouth and acts in very much the same way as the pepsin of the gastric juice, except that it does not require the presence of acid.

A consideration of the question of etiology would seem to suggest that the prevalence of caries in modern races is due to the soft character of the food, and the increase in the use of refined carbohydrates, some of which undergo rapid fermentation. It is probable that defective calcification of the enamel influences the incidence of caries in some cases.

So far then, as our present knowledge goes, dental caries is started by solution of the inorganic salts of enamel by acid derived from carbohydrate fermentation ; variations in form and structure and inefficient functioning predispose to its incidence, as also do extrinsic factors such as certain food idiosyncrasies and occupations ; it is probable that there are additional predisposing factors, of which variations in the composition or reaction of saliva such as may be brought about by a condition of acidosis or the action of radium on the salivary glands may be important ones.

(G) SYMPTOMS.

Pain in the region of the affected tooth may occur. If the caries is on a surface free to the tongue, the patient will be conscious of a cavity. In many instances, the lodgment of food between the teeth is the first symptom noticed. The pain is due to irritation of the non-medullated nerve elements contained in the dentinal fibrils, and will vary according to the situation and extent of the cavity. On occluding surfaces, the fibrils being exposed, changes of temperature or the introduction of irritant substances into the cavity may produce pain which increases in severity as the cavity becomes larger. In cavities tucked away under the gum or on approximal surfaces, pain is often not felt until the pulp is involved. The degree of pain varies with different temperaments, and in the same individual at different periods. Individuals of a nervous temperament suffer more than those of a lymphatic temperament. In a few individuals, caries gives rise to no pain.

(H) DIAGNOSIS OF DENTAL CARIES.[1]

The diagnosis of dental caries in the early stages is often difficult, but when the condition is well established it is simple as, in addition to decalcification of the dentine, there is extensive destruction of the enamel. When the disease has advanced to this stage there is usually some involvement of the dental pulp, which becomes infected and finally destroyed if decalcification takes place more rapidly than the formation of secondary dentine.

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[1] For the details of the diagnosis of dental caries by radiographic means we are indebted to Claude G. Colyer.

In order to repair teeth before serious damage is done, it is essential to diagnose the condition as early as possible, and this is best done by means of periodic examinations which should be made at least every six months.

Caries is usually to be found in one or all of the following situations :—

(1) In the fissures on the occlusal surfaces.
(2) Around the necks of the teeth above the gum margin.
(3) Interstitially immediately below contact points.

In the first place, all fissures should be carefully examined with a finely pointed probe ; if this is found to enter any fissure or crack it is safe to assume that caries has commenced.

The white areas around the necks of the teeth and on the approximal surfaces are usually due to incipient caries having caused some disintegration of the enamel. Caries is most commonly found where teeth are actually in contact with each other, and may be detected by one of the following methods :—

(1) A curved probe, or one bent at right angles at the end will, if passed between the teeth, engage in any depression on the surface of the enamel.

(2) Floss silk passed between the teeth will become frayed or broken where there is some destruction of enamel.

(3) Changes in colour (a loss of translucency) may often be noticed when the teeth are examined with a bright light ; a whitish appearance on the occlusal surface near the contact point is due to decalcification of the dentine beneath the enamel. If the anterior teeth are examined by transmitted light any caries areas will appear as dark shadows.

(4) The most reliable method of detecting early caries near the contact point is by means of a radiographic examination.

This method of radiographic examination was first described by Rapier of the United States, and consists of the exposure of films placed on the lingual side of the teeth so that the crowns of both maxillary and mandibular teeth appear on the same film. While the exposures are being made the special films, which are made for the purpose, are kept in position close to the lingual side of the teeth by a tag attached to the sensitive side of the packet containing the film, or if ordinary films are used they should be placed in a special holder (fig. 438) which is held firmly between the teeth; a handle or director being attached to it at

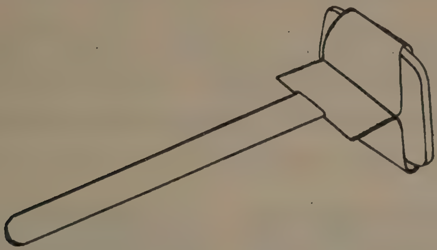

FIG. 438.

right angles so that the rays can easily be directed along it on to the tooth as well as the film (fig. 439). It is essential to direct the rays carefully along the handle so as to avoid overlapping of the teeth which would obscure any rarefactive changes on the surface of the enamel.

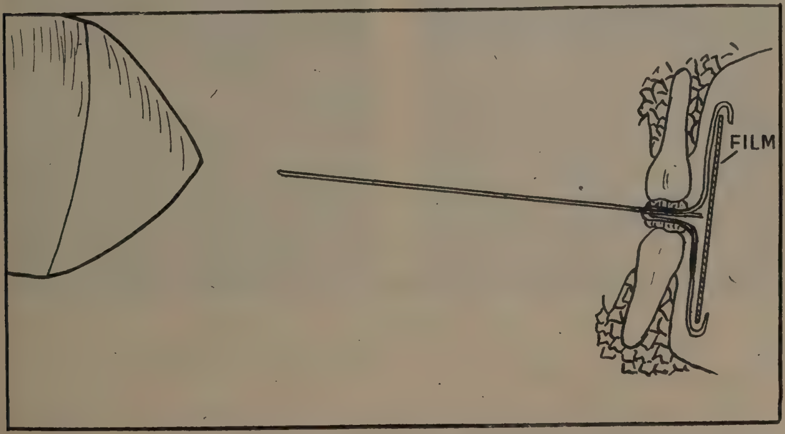

FIG. 439.

The film holder (see fig. 438) is made of aluminium, which affords but little obstruction to the X-rays and consists of a slide into which the film is inserted, a flat plate covered with rubber to be held between the teeth and a handle along which the X-rays are directed. The holder being rigid, the insertion of the film between the tongue and the teeth is simplified. By means of the handle the film is pulled as close to the teeth as possible. In order to do this successfully the corners of the film should be bent, so that they do not impinge on the mucous membrane and cause pain. (See fig. 439.)

The films produced in this way will appear as in fig. 440, and show distinctly the enamel differentiated from the dentine and the size of the pulp chamber. It will be noticed that the surface of the normal enamel is regular in outline and of uniform density as the distal surface of $\begin{array}{r|l} & \\ \hline 4 & 4 \end{array}$ and the mesial surface of $\underline{|\,4}$. Should the outline of the enamel be broken or the density be altered in places, as in the approximate surface of $\underline{54\,|}$ it is evidence that caries has commenced. In the same figure $\overline{76\,|}$ show a more advanced condition, and one which is very favourable for treatment, as decalcification of the dentine has commenced under the enamel; whereas in $\underline{5\,|}$ caries is so extensive that complete removal of the softened dentine would necessitate exposure of the pulp.

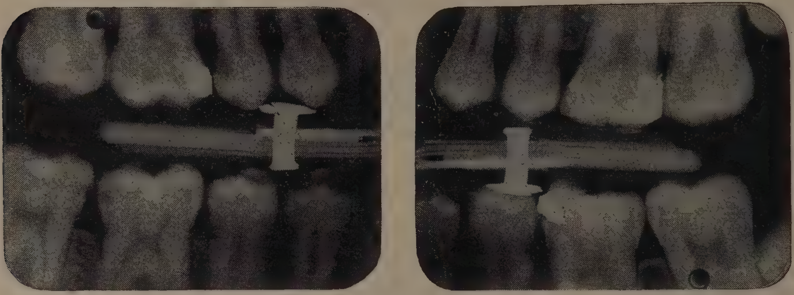

FIG. 440.

Well-established caries is present in: $\begin{array}{r|l} {}^{1}5 & \\ \hline 7_{11}6 & {}_{1}7 \end{array}$. Early caries in: $\begin{array}{r|l} {}^{1}65^{11}4 & \\ \hline 6_{11}5 & {}_{1}6 \end{array}$

All translucent areas appearing in the dentine contiguous with irregularities in outline of the enamel surface are due to caries and, unless the teeth are isolated, require treatment.

Rarefied areas under fillings are usually due to recurrent caries and require immediate treatment, as $\overline{|\,6}$ mesial in fig. 440. One note of warning: synthetic fillings resemble caries in their radiographic appearance, but are usually more clearly defined.

A typical case before and after treatment is shown in fig. 441. The extent of the caries is well shown. It is interesting to observe how much more extensive the decalcification is than would be imagined from the radiographic appearances; especially note $\frac{\,|\,}{75\,|\,7}$.

(I) SPONTANEOUS ARREST OF CARIES.

The spontaneous arrest of caries occurs in both deciduous and permanent teeth, though far more commonly in the former.

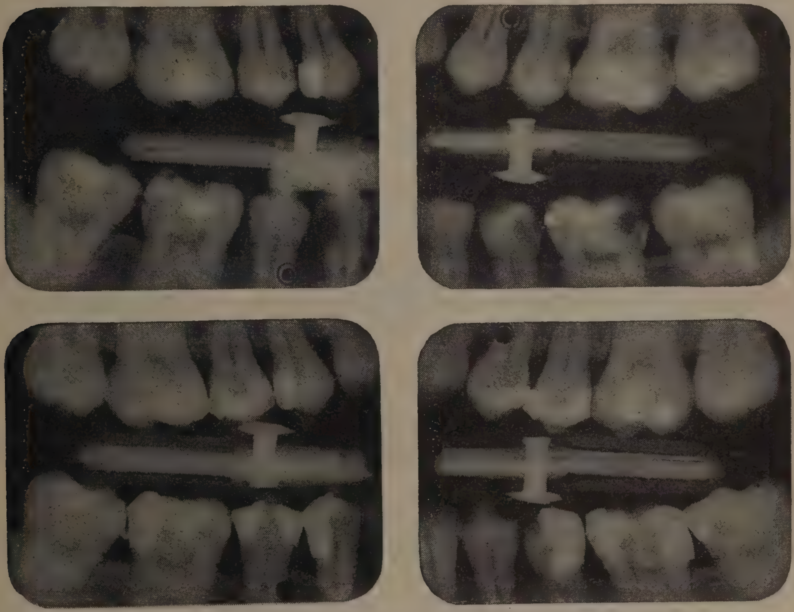

FIG. 441.

Shows the following teeth before and after treatment: $\frac{6^{11}5^{1}\quad|\;4^{11}5^{11}6}{7_{11}6_{11}5_{11}4\;|\;5_{11}6_{11}7}$

It occurs commonly in the deciduous molars; in the permanent dentition it is most often seen in hypoplasic permanent first molars, but it may occur in any tooth.

The factors necessary for its incidence in perfection are: more or less extensive superficial coronal or occlusal caries, the absence of pain, efficient functioning, and a live pulp; it may occur, but comparatively rarely, in pulpless teeth and stumps, but only when they are painless in use; the process is then entirely one of denudation or the wearing away of the carious portion, and there is then no vital reaction (hypercalcification) of the remaining dentine. Its occurrence is, perhaps, more readily explained as it occurs in permanent first molars of defective formation.

When such teeth become carious, caries often commences at many points almost simultaneously. The force of mastication then breaks in the undermined occlusal enamel extensively or entirely at these many points, so that a large area of superficially carious dentine is exposed; being superficial and therefore some distance from the pulp the tooth is not painful, and mastication may still be performed with it. This act wears away the decalcified and infected surface dentine and the exposed dentine surface being a large one is not very retentive of food particles; in other words, it is more or less self-cleansing, but being softer than normal (because a veneer of partly decalcified dentine is still present) it readily becomes stained by the food, stains, etc., to which it is exposed till it ultimately becomes dark brown or nearly black.

In the meantime the tooth pulp, in the case of live teeth being exposed to unusual thermal and masticatory stimuli, reacts so as to cause calcification of all dentine fibrils which lead on to the exposed surface, so that ultimately they become entirely calcified and their dentinal tubes are occluded. The proof of this latter lies in the fact that as wear of the dentine progresses the stained portion is worn away and unstained (because impermeable) dentine appears on the surface; this latter appearance is more often seen on the deciduous molars, where it is very common, than in the permanent teeth, though it is occasionally seen in these also.

It occurs so much more often in deciduous molars because of the well-known clinical phenomenon that the dentine of children's teeth is nothing like so sensitive as is that of adults, so that in spite of extensive superficial caries the teeth function well. Partly for this reason the process is far more often seen in perfection in deciduous molars. In permanent first molars, the destruction of the occlusal enamel places these teeth on a rather lower level than before, so that stagnation areas may be produced in the angle of junction with the crowns of the teeth anterior and posterior to them, so that the self-cleansing action of mastication is not so effective. The result is that the spontaneous arrest of caries in these teeth is often imperfect in that it is only partial, and active caries often persists anteriorly and posteriorly.

The surface of the dentine in cases where the spontaneous arrest is well established is usually polished by use.

Histologically, the dentine tubes appear uninfected when the process is well established (fig. 442).

Among the new admissions having extensive caries of deciduous teeth that one sees in a large residential home, over half show some stage of the spontaneous arrest of caries, and sometimes in many teeth.

The researches of M. Mellanby have led her to conclude that administration of the calcifying vitamin D in such form as ergosterol or cod-liver oil may bring about the spontaneous arrest of caries ; there is, however, at present little evidence to support this conclusion. In one case under notice a girl, aged fifteen, had for the previous eight years been treated with daily doses of cod-liver oil and extra milk (pasteurised) during school term (nine months in the year), but during the same period twenty-three fresh carious cavities appeared in her teeth ; not only was there no spontaneous arrest of caries but apparently no prevention ; several other similar but less striking cases have also been noticed.

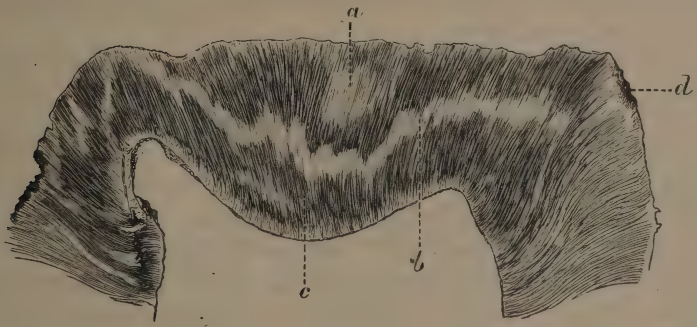

FIG. 442.—(*a*) Portion of tooth in which caries is becoming arrested ; (*b*) translucent zone ; (*c*) normal dentine ; (*d*) a softened patch of dentine showing micro-organisms.

One may conclude by saying that, when the conditions necessary for its incidence are present, spontaneous arrest of caries in 'live' teeth is usually brought about by three factors : (i) Increased activity of a healthy tooth pulp in response to stimulus, causing hypercalcification of dentine (or enamel, as shown by Howard Mummery (see fig. 408)) ; (ii) a fully adequate diet, which will assist the fulfilment of factor (i); and (iii) denudation of the softened dentine (or enamel) by functional activity; this latter being caused either by attrition with an opposing tooth, or food friction, or both.

In pulpless teeth it can obviously only be due to the process of denudation; but to avoid confusion it must be remembered that the tooth pulp may become exposed and die as a result of active caries in one part of a tooth *after* it has produced the hypercalcification of 'arrested' caries on another part (usually the occlusal aspect), and indeed this frequently happens.

Assisting the Process.—On occasion it is highly desirable to assist, or even bring about, the spontaneous arrest of caries; both are easily done in suitable cases and it is an invaluable aid, particularly in the treatment of extensive caries of many deciduous teeth in the same mouth.

Treatment consists in stoning away any prominent enamel edges, which cause stagnation areas at their angle of junction with the dentine; the cavity must be made non-retentive, and, as far as possible, part of the occlusal surface of the tooth, and the soft infected surface dentine removed; the exposed dentine is then sterilised by treating it several times with silver nitrate applications, an albuminate of silver is formed with whatever organic matter the silver salt comes into contact, and sensitiveness of the dentine, if present, is allayed. A fully adequate diet will always assist this process.

CHAPTER XI

Prophylaxis and Treatment of Caries.

Prophylaxis—Remedial Treatment—The Operation of Filling—Hypersensitive Dentine—The Operation of Excision—The use of Drugs in the Treatment of Caries—The Operation of Crowning.

The treatment of caries may be divided into

(A) Prophylactic.

(B) Remedial.

(A) PROPHYLAXIS.

Rational treatment of caries, like the rational treatment of any other disease, must depend upon knowledge of the etiology of the disease. When the cause of a disease can be traced its eradication can generally be quickly accomplished by the adoption of measures for the removal of that cause.

Although, during recent years, advances have been made in our knowledge of the etiology of dental caries, we cannot yet claim that all the factors influencing the disease are entirely known.

In discussing the cause of caries, attention was drawn to certain matters which appear to have a special bearing on the increase of caries, namely, the feeding in infancy, and the physical character of the food-stuffs, etc. A consideration of these matters seems to indicate that preventive treatment should be based on the following factors :—

(*a*) Pre-natal nutrition.

(*b*) Feeding and care in infancy and childhood : the chemical and physical nature of foods and the manner of taking food.

(*c*) Feeding and care after dental development is complete.

(a) Pre-natal Nutrition.—The teeth are part of the body and not separate entities ; it is obvious that certain materials are required to build that body in perfection, both before and after birth, and if these materials are inadequate, or not available in assimilable form at the time they are needed, either the body, including the teeth, will be faultily made or the child may die. Before the child is born it is a parasite on its autosite mother, and if these building materials are not then fully available, the mother's body will suffer first as the parasite grows at its mother's expense, but this does not apply to the mother's teeth, which are already completed, though it may affect the cells and metabolism of her body so that they in turn affect her oral structures, secretions, and teeth.

In turn, this shortage affects the child, it can interfere with the formation of dental tissues (see p. 59), it may possibly hinder the development of dental formative cells so that even if dietary perfection is attained later, they may not then be able to function in perfection. The occurrence of structural faults in the deciduous teeth is evidence, either that the building materials have not been available, or that something has interfered with the proper functioning of the formative cells, though conceivably this something is not necessarily a dietary deficiency.

Though the foregoing is logically true, there is little other reason to suppose that maternal food deficiencies affect the structure of teeth, particularly the permanent teeth; indeed, there is no evidence that the formative cells of the permanent teeth are affected by maternal influences save in the transmission of infections and hereditary variations, and investigation in the United States [1] has shown that 85 per cent. of the total calcification even of the deciduous teeth takes place after birth.

(b) Feeding in Infancy and Childhood.—There can be no second thought that the ideal food in the earliest part of a child's life is its mother's milk. It is true that *breast-feeding* cannot always be performed. Often hand-feeding is substituted, not so much from inability of the mother as from a desire on the part of the mother to escape the ties which the duty of breast-feeding involves. When breast-feeding is impracticable, every care should be taken to ensure that the hand-feeding is carried out in a proper manner.

In the act of suckling, a child does not merely take the mother's nipple into its mouth, but a mouthful of breast; so that the nipple is well to the back of the mouth; doubtless the efforts put forth by the jaws, tongue, and cheeks on the breast in the child's efforts to obtain nourishment account for the greater development of the jaws in width found in breast-fed children and, as they are obtaining their natural diet (and no adventitious refined or readily fermentable carbohydrate), accounts for the lesser incidence of dental caries in the teeth of such children.

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[1] Hess, A. F., Lewis, J. M., and Roman, B. *Dental Cosmos*, 1932, 74, 1060.

Milk contains everything that the helpless and edentulous mammalian young need in the way of chemicals and vitamins till they begin to get teeth, when additions should be made, as, for instance in human children, there is found to be a shortage of iron about that time. At the same time, if the maternal diet is deficient in chemicals and vitamins the child also may be affected ; rickets, for instance, has occurred in an entirely breast-fed baby[1] whose mother's diet was deficient in many necessaries.

It is worthy of note that primitive races to whom the use of cow's milk in the feeding of infants was unknown, such as the Maoris, Esquimaux and North American Indians, and also the Japanese, and who had no other means of feeding babies, used to suckle them for six years or more, and a reference to J. R. Mummery's work on pp. 304 and 365, and H. Pickerill's work, will show that the first three of these races had the least-known incidences of caries in man ; the incidence in the Japanese at that time is unknown. H. Stobie has stated concerning Kaffir and Zulu children, who are suckled from two and a half to three years, that he has never seen caries in their deciduous teeth, though their mouths were as dirty with sticky adherent carbohydrate as any he has seen.

In those races who practised prolonged suckling, if a child's mother died and a relative was not able to wet-nurse it, the child was destroyed, as there was no other means of keeping it alive. Incidentally, suckling is Nature's method of birth-control to a large extent by inhibiting ovulation.

It should also be realised that before the advent of proprietary infant foods physiological suckling of from 2½ to 3 years was practised in this country, and as evidence one may quote from *Romeo and Juliet*, in which Shakespeare (A.D. 1564–1616) puts the following words into the mouth of Juliet's nurse (Act 1, Scene iii) :

> ' On Lammas-eve at night shall she be fourteen ;
> That shall she, marry ; I remember it well.
> 'Tis since the earthquake now eleven years ;
> And she was wean'd,—I never shall forget it,—
> For I had then laid wormwood to my dug,'

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[1] *Lancet*, 1933, ccxxv, ii, 757.

Prolonged suckling is impracticable, unnecessary, and out of keeping with the present knowledge of child requirements and the conventions of civilised society, and after a period of nine or ten months' maternal feeding, cows' milk, or preparations of it, are usually substituted.

In 1932 one of us published[1] the clinical findings in a large number of children who had been brought up largely on raw cows' milk. The initial observation was made on a group of 750 boys, when, owing to the somewhat sudden cessation of 'return' cases of caries, tabulation of all boys who had been under this regime prior to erupting their permanent first molars was made; as part of their diet they had a pint of milk to drink daily, and were as follows :—

COMMENCING RAW MILK BEFORE THE SIXTH BIRTHDAY ANNIVERSARY, PRESENT AGES RANGE FROM 7 YEARS TO 10 YEARS 9 MONTHS.

Number of boys.	Average age at commencement.	Average age now.	Incidence of caries in all permanent teeth.
28	4 years 8 months	8 years 10 months	2 carious maxillary permanent first molars in 1 boy

There were 4 hypoplasias (2 marked) but with no caries, and 1 boy had stained fissures in 2 teeth.

Nearly all the boys in this institution are subnormal and the one who had the carious molars had spent several years continuously in hospital.

It seemed then that if milk was at all an etiological factor in producing this lessened incidence of caries, children who had been brought up on it should show a very small incidence, and in other branches of the same institution, to which weakling babies are sent, a total of forty children was found who had been started on the raw milk regime at an average age of four and a half months and were, at examination, of an average age of four years, and these forty had *no incidence of caries at all.*

Most of them had one or two sweets as a routine after their midday meal, and oatmeal porridge six days out of seven. They also had raw egg yolk ; and egg yolk and milk constitute Nature's fundamental sources of complete nourishment for helpless and growing vertebrates.

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[1] *Proc. Roy. Soc. Med.* (Sec. of Odontology), 1932, 25, 11. (E. S.)

The above appears to be evidence that diet during the pre-eruptive period influences the incidence of caries in man; prior work had been attempts to influence already erupted teeth, and usually under observation for periods of a few months only.

The above, and much other actual and circumstantial evidence, prompted the statement that milk 'seemed to have a profound influence on the development and calcification of the teeth of man, in that it conferred on them some immunity to dental caries.' On this and other evidence several authoritative writers took it that the value of milk as a preventive of caries had been demonstrated, and said so,[1, 2, 3] but any claim that dental caries can be prevented under any circumstances by a particular item of diet seems to be absurd on the face of it.

In Tristan da Cunha, where raw milk is a staple item of diet, we know that for very many years there has been practically no caries; even in the eight-year period, 1923–31, when they had [4] up to two tons of sugar and three and a half tons of flour per annum, no difference was found, but since then a still greater increment of carbohydrate seems to be making the scale turn, though not to any great extent yet.

At Pitcairn Island,[5] where the inhabitants are of mixed race ancestry (as in Tristan), caries is rampant, and they lose most of their front teeth from caries while quite young; other teeth may or may not be carious or absent. Their diets are very similar to those of Tristan except that there is no milk available on the island save maternal milk, and occasional tinned milk as a luxury; sugar cane is also grown there, 'syrup made from its juice being used instead of sugar.'

At Christ's Hospital, Horsham, there are 800 boys; since 1903 a daily ration of raw milk has been issued to all boys up to 12 years of age. In 1903 68 per cent. boys showed fresh caries, averaging 2·8 cavities each; the milk ration was increased in 1921, and again later, so that by 1933 they were getting 0·78 pint daily, and about this time the annual incidence of fresh caries had dropped (in 1931) to 1·5 cavities each in 37 per cent. boys only. Prior to the first increase in 1921 the annual incidence for twenty-two years had been 2 to 2·8 cavities per head in such

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[1] *Bulletin* 5. The Hannah Dairy Research Institute, 1933, 71.

[2] Frazer, A. C. *Brit. Dent. Journ.*, 1933, 54, 401.

[3] *The Dental Surgeon*, Editorial, July, 1932.

[4] Gane, D. 'Tristan da Cunha,' 1932, 154.

[5] *Brit. Med. Journ.*, 1932, ii, 516.

as had any fresh caries. Putting it in other words, the yearly fresh caries in the School has dropped from 1,524 cavities in 1903 to 444 in 1931, and that is where milk is not started till comparatively late in childhood. Other dietary improvements were also initiated in the post-war period, but even so, the Medical Officer to the schools wrote :[1] ‘ If the dental figures are examined in relation to the amount of milk consumed per head a close correlation can be traced between the gradual improvement in general condition and the decreased incidence of caries.’

In the Annual Report of the School Medical Officer to the County Council of East Sussex for 1933 are the details of an inquiry into the circumstances of all children between 12 and 13 years of age found with perfect dentitions. His three school dentists returned a total of 52 such children. Details furnished by the County Medical Officer of Health and collected by his District Nursing Superintendents and School Nurses showed that 32 of these children were still at this age drinking raw milk daily, ‘ plenty ’ in quite a number of cases. Other details of these 52 are of interest : 17 never brushed their teeth, only 5 did not eat sweets, some of the others are described as ‘ always ’ eating them or eating a ‘ good many.’ Two of them had 10 and 11 brothers and sisters respectively with no carious teeth in any of them ; 31 were of ‘ good general physique,’ and 20 ‘ average,’ 12 were of ‘ exceptionally good ’ domestic circumstances and 31 ‘ average.’

A similar return made in a distressed area in the North of England in the same year only returned five such children as free from caries ; three of these were returned as still at this age having raw milk daily.

It is of interest to compare children who have been on pasteurised milk. Since March 1932, 1,400 girls in a branch of the same institution as the boys tabulated on p. 384 have had a daily ration of three-quarters of a pint of pasteurised milk : this may have been used in tea, cocoa, milk puddings, etc., or taken as milk. There are 39 children who were before their sixth birthday anniversary when this was started and have been there continuously since, and whose average age is now (December 1937) 9 years 1 month ; they now show 25 carious teeth in 12 children as follows :—

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[1] Friend, G. E. ‘ The Schoolboy, a Study of his Nutrition, Physical Development and Health.’ Cambridge, 1935.

COMMENCING PASTEURISED MILK BEFORE THE SIXTH BIRTHDAY ANNIVERSARY, PRESENT AGES RANGE FROM 6 YEARS 5 MONTHS TO 11 YEARS 8 MONTHS

Number of children.	Average age at commencement.	Average age now.	Incidence of caries in all permanent teeth.
39	4 years 4 months	9 years 1 month	25 carious in 12 children

There were 4 hypoplasias (2 marked) but with no caries; and 3 had stained fissures in 1 tooth each, but 2 of these had otherwise no caries.

Though it is obvious that these are far better than the average school clinic child of this country, they do not compare well with the boys of similar ages quoted as on raw milk in 1932 (see p. 384).

If milk is used raw it should necessarily be bacteriologically safe; for this purpose 'certified' milk from tuberculin-tested animals which have also been *B. abortus* tested is best, as bovine tubercle is still a very common disease in this country and communicable by milk to children. In this country *B. abortus* infection is very rare in children, though occasional in adults; it is also trivial in children, also it is not always communicated by drinking milk. One of the virtues of raw milk is that it has to be used fresh, and perhaps the gravest objection to pasteurised milk is that on keeping the residuum of putrefactive and other bacteria multiplies enormously without obvious change in the milk; not so long ago an instance of pasteurised milk was recorded [1] which on sale contained 178 million organisms per cubic centimetre of which 29 millions were still living. After serving as a nutrient medium for them it had probably not much food value for children. It is possible that such loss of nutritional value on keeping may be a major factor in the lessened dental benefit which pasteurised milk seems to show.

That changes take place in the constitution of milk on heating is well known: vitamin C, for instance, is destroyed, and there is also a change in the diffusible calcium; these and other changes are regarded differently by different people, some regarding them as of no importance and others as lessening the nutritive value of milk apart altogether from its vitamin content.

As the teeth come into position all foods should be such as compel mastication or such as the child may drink. The use of such foods as can be neither masticated nor drunk are heartily disliked by the great majority of children with normal teeth. Bread and milk, milk puddings, and porridge are among such foods, and the tendency is for the child to squash them with its tongue against its palate and swallow them—all of them contain refined carbohydrate in a sticky and sometimes readily fermentable form. Mastication promotes a free flow of saliva which assists in cleaning the teeth and removing starchy carbohydrates besides maintaining physiological activity of the periodontal membrane, gums, teeth, and musculature of the jaws.

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[1] Dockeray, G. C. *Brit. Med Journ.*, 1933, 1, 337.

Foods too should be related to the child's chemical and vitamin requirements, and in so far as possible of an unrefined nature, particularly at the end of a meal.

It is commonly and correctly quoted that the teeth of institutional children are better than those of the same social class outside institutions ; this is usually, but questionably, ascribed to the less amount of refined carbohydrate and sweets which such children get, as one finds on enquiry that they get quite an appreciable amount of both, and perhaps more than middle class children did in pre-war days and earlier ; it is very likely that milk, which practically all young institutional children always have had, may have influenced this lesser incidence of caries.

It is not inapt to compare the building of a tooth to the building of a house ; skilled workmen (efficiently formed and functioning cells) using the best materials will produce the perfect house (tooth) on an ideal foundation (parodontal tissues), and poor workmen using such indifferent and substitute materials as they can obtain will produce a jerry-built house with faulty foundations. Yet when first finished both may look identical ; if one can imagine the houses as being reduced to the size of teeth, histological and chemical examination might show but little difference, but there can be no question as to which will better withstand the stresses and actions of time and adverse elements, though both are acted on by them. So also with the teeth and their attachments.

The Personal Factor.—Another fact which tends to confuse and produce exceptional cases is that the assimilable or metabolic capacities of different children vary widely, and this, in its more moderate variations, probably occurs more often than we are apt to credit. In 1932 three cases of twins were recorded ;[1]

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[1] *Brit. Med. Journ.*, 1932, 1, 769, 862 and 912.

in each case the feeding of the twins had been identical, yet at about five years of age one of each pair was a normal healthy child and the other suffered from pronounced rickets, and rickets, a disease largely due to dietary deficiency, can profoundly affect the teeth. At present we are totally unable to assess the importance or frequency of this variation in metabolic capacity, nor do we know why it occurs. We might well call it the ‘Personal Factor.’

Granting that perfect dietary during the tooth-formation period will build the perfect tooth, the question naturally arises: Will caries then be prevented? The correct answer seems to be that so long as enamel is made of materials soluble in acids, and so long as decalcifying substances can come to a state of stasis on or around teeth, nothing can prevent caries, but it seems certain that the early date of its commencement, and its rapidity, will depend partly on structure and shape, and that both structure and shape are influenced by pre-eruptive nutrition.

It must be remembered that many chemicals as well as vitamins are necessary for the proper formation of teeth; calcium and phosphorus and vitamins C and D are, we know, necessary among many other things, and since the absence of any one of these may make the forming of a tooth, and even life, impossible, one cannot say that one of these items is of greater importance than the others, as all are needed in sufficient quantity and assimilable form at the right time.

In the paper on milk already referred to [1] it is stated: ‘There seem also to be physical differences of a protective nature developed . . . deep and sharp fissures and pits appear to be absent from the occlusal surfaces and other aspects where they occasionally occur, and the cusps are less sharp and pointed; these appearances are best seen in the permanent first molars, and, at their maximum, make them almost typically bunodont teeth; possibly these changed appearances are brought about by increased bulk in the enamel.’ One feels convinced that such changes are dietetic in origin, and in further support of this view attention is drawn to those pits on the buccal aspect of mandibular molars which were seen extremely commonly in pre-war days but do not appear so commonly now (fig. 443). Comparable pits also occurred on the palatal and buccal aspect of maxillary molars, not quite so commonly perhaps, but one hardly ever sees them now. These faults appear to be comparable with other trophic structural defects such as are dealt with on p. 54 *et seq.* Inquiry among dentists having present-day experience of children in private practice and school clinics confirms the comparative rarity of these pits in the former as compared with the latter type of child. These improvements in the formation of enamel and also the decreased incidence and extent of hypoplasia in teeth are, it seems, probably the results of teaching at maternity and child-welfare clinics and the greater knowledge of dietetic values in infant feeding.

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[1] *Proc. Roy. Soc. Med.* (Sec. of Odontology), 1932, 25, 11. (E. S.)

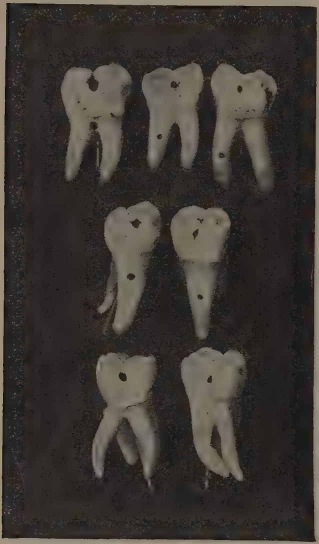

FIG. 443.—A (top row). Permanent mandibular molars showing the pit on the buccal aspect, believed to be disappearing as improvement in the nourishment of children in the early years of life is brought about. B and C (second and third rows). Comparable pits on the palatal and buccal aspects respectively of permanent maxillary molars, believed to be disappearing as in A. (E. S.) (From *Brit. Dent. Journ.*)

There is little doubt that of the carbohydrate foods, sugars are the most harmful, especially when taken between meals, and particularly the monosaccharide glucose, now so frequently used and prescribed, as organisms can act on it directly in the mouth with the production of acid. *'Sweets' as popularly understood should be forbidden.* As carbohydrates are now known to be the source from which lactic acid is derived, it is advisable so to arrange a meal that the last food-stuff taken is neither a sugar nor a starch. A meal should, if possible, finish with fruit or cheese. Fruit especially is beneficial, as it is slightly acid and stimulates the flow of saliva. Meals should be given at regular intervals. If the salivary secretion is constantly being stimulated by incessant 'nibbling' at food, it is probably robbed of its physiological properties and the supply required for the regular meals is depleted and probably altered in character.

A functional mouth is essential if caries is to be prevented. There are two important factors militating against a functional mouth: one is mouth-breathing and the other the presence of tender teeth. Mouth-breathing, as explained on p. 507 *et seq.*, leads to a persistent marginal gingivitis which aids the retention of food around the teeth besides being a source of toxic absorption. It is very important therefore that any condition causing nasal obstruction should be removed in order to induce correct breathing. Tender teeth hinder mastication, and without proper mastication the natural cleaning of the teeth and nourishment of the child is interfered with. The mouth, then, must be freed of all tender teeth; this in itself is conservative of neighbouring teeth by allowing them to function more efficiently.

The proper cleansing of the teeth after meals is a most important prophylactic measure; obviously it is best if this be brought about by physiological means, but if the conventions of civilisation are adhered to artificial means are necessary. Under these circumstances the teeth should be cleaned after every meal, but specially after the last meal of the day. The tooth-brush should be small and contain fewer bristles than the brushes usually sold; the bristles should be of different lengths, so as to permit of their passing more easily into the interstices of the teeth. The following instructions for cleaning the teeth are of use:—

(1) Use a small tooth-brush with soft but stiff bristles, such as badger hair. Use common salt or a little soap and some precipitated chalk.

(2) Brush all surfaces of the teeth thoroughly in all directions, especially the back ones.

(3) Rinse the mouth thoroughly with clean water.

(4) Clean the teeth immediately **before going to bed.** Take no food of any sort afterwards.

In mouths **where caries is progressing rapidly,** it is a useful measure to swab the mouth with an alkali the last thing before going to rest, for example:—

Mag. carb. levis	ʒiv.
Aq. rosæ	℥vj.
Aquam	ad ℥xij.

Misce. Shake before using.

After cleansing the teeth, a tablespoonful of the above should be taken into the mouth and sluiced between the teeth.

In cases of acute fevers, or any other condition causing helplessness, the patient's teeth should be cleaned by the nurse.

(c) Feeding after dental development is complete.—The chemical and physical nature of food requirements after dental development is complete are similar in principle to those of childhood ; for the observant student there can be no need for reiteration—chemical and vitamin sufficiency, and the use of unrefined and self-cleansing foods particularly at the end of a meal, are the ideals one should seek to maintain oral and dental cleanliness in the ways already indicated.

Patients wearing dentures should be warned of the danger of neglecting to cleanse both the mouth and the dentures, especially where there are bands or clasps. Dentures cause caries only in so far as they assist in retaining food around the teeth.

In mouths, showing a tendency to approximal caries of the anterior teeth, isolation should be obtained by the removal of the first or second premolars or the permanent first molars (see p. 211). Isolation of the incisors and canines is a most valuable method of preventing caries between these teeth.

(B) REMEDIAL TREATMENT.

The remedial treatment may be considered under the following headings :—

(1) The operation of filling.
(2) The operation of excision.
(3) The use of drugs.
(4) The adaptation of artificial crowns.
(5) The operation of extraction.

(1) THE OPERATION OF FILLING.

At the present day the methods of cavity preparation and the variety and methods of inserting fillings are ever increasing. The authors consider that this (' operative ') branch of Dental Surgery may be excluded from the present edition, as its scope has now become so large and its technique so varied and intricate that it is better to be dealt with in text-books dealing solely with this subject.

Hypersensitive Dentine.

The preparation of cavities for filling is at times hampered by hypersensitive dentine. The area of hypersensitiveness frequently varies, one portion of the tooth being distinctly more painful than another. The portions of dentine immediately beneath the enamel and at the neck of the tooth are usually the most sensitive.

The pain can be greatly alleviated by keeping the cavity dry during excavation, and by using sharp instruments with a decided cutting and not scraping action.

Treatment :—

Dehydration.—The application of hot air in the form of blasts from a suitable syringe greatly reduces the sensitiveness. The hot air may cause a little pain, but if it is applied with tact and care the pain can usually be avoided. The hot air dehydrates the dentine. If absolute alcohol is applied before the hot air, the dehydration is increased, but the treatment is more painful, though the pain is lessened if the alcohol is used warm.

The use of a continuous cold water jet into the carious cavity while instrumentation with a bur is in progress is invaluable as a desensitiser of dentine. Cold lowers the conductivity of nerves and the effect is probably brought about in this manner, though doubtless there is some additional psychic effect in many cases. The jet should be on the actual point where the bur is being used and should not be allowed to pool around the tooth ; its effects are not so constant or good in people over fifty, and are of no value whatever when using an excavator.

The Local Application of Drugs.—Strong carbolic acid may be allowed to remain in the cavity for a few minutes and the dentine dried with hot air. This remedy is not very efficient if immediate relief is desired, but if carbolic acid is sealed in the cavity for two or three days a decided obtunding of the dentine is obtained.

Chloride of zinc is a useful drug, but it must be used with caution when the pulp is near. The cavity must be isolated and a small piece of chloride of zinc in the solid state allowed to deliquesce. The application causes pain, but the pain rapidly passes away.

Nitrate of silver causes unsightly discoloration and can only be applied to the posterior teeth. It is slow in its action. In superficial cavities, nitrate of silver is applied in a solid state fused on the end of a metal instrument, and should be rubbed over the sensitive surface. The tooth is then left for one or two days, when the dentine becomes less sensitive. In deeper cavities, a little of the powdered drug may be sealed in.

Arsenious acid has been used for the desensitising of dentine ;

it acts by destroying the fibrils, it does not coagulate tissues, and its use for this purpose is dangerous as its action is progressive and may ultimately destroy the tooth pulp unless entirely removed after application, and this is a difficult thing to do efficiently.

If it is used it should only be for shallow and superficial cavities and not applied for more than two hours.

Where it is intended to use metal fillings and the dentine is sensitive, the best course is to insert an oxy-phosphate filling and allow it to remain for about six months. In some instances the tooth substance is so sensitive that it is almost impossible to remove any of the carious tissue. A filling of oxy-sulphate of zinc, if left in place for one or two weeks, will allay the sensitiveness sufficiently to allow of the preparation of the cavity. For posterior teeth which are very sensitive, the application of nitrate of silver may be substituted for the oxy-sulphate.

Paraform, a polymer of formic aldehyde, is also used for this purpose ; it is used diluted with twenty times its weight of zinc oxide or other inert substance made into a paste with gum water. It may be left in one or two days. It acts by virtue of its formic aldehyde content and coagulates the dentinal fibrils ; if placed near the pulp it will cause pain, and may destroy its vitality.

Local and Regional Anæsthesia.—In cases where the teeth are extremely sensitive and the local application of drugs fails to relieve the pain, local anæsthesia should be produced by the use of novocain (see Chapter XXXVI). The tissues may also be rendered insensitive by the use of freezing agents.

(2) THE OPERATION OF EXCISION.

In the operation of excision, the diseased part is stoned and disked away and the surface polished. With the permanent teeth it is applicable in very few cases, the most suitable being the approximal surfaces of the molars and premolars when the caries is quite superficial and the surfaces are exposed, but it may be of considerable value in the treatment of the deciduous teeth, particularly the molars, especially in conjunction with the use of silver nitrate.

(3) THE USE OF DRUGS.

The progress of caries may be considerably retarded and often arrested by the application of certain drugs, the most useful of which is nitrate of silver. Drugs can be employed to their greatest advantage for superficial caries.

The **nitrate of silver** is applied in the form of a saturated solution, or, still better, the solid drug is rubbed over the surface. Two or three applications, at intervals of about a week, will effect a considerable hardening of the surface. The nitrate of silver precipitates the albumin in the tubules and forms an albuminate of silver which is a powerful antiseptic. The discoloration produced is an objection to its use on the anterior teeth. In cases of general softening of the cement often seen in patients whose gums have receded, the application of nitrate of silver to the back teeth and **chloride of zinc** to the anterior teeth, with the regular use of **alcohol locally,** will retard and often arrest the condition. The alcohol should be used as follows :—

The teeth are dried and absolute alcohol applied to the roots either on a brush or cotton-wool, the saliva being kept away for about two minutes. The alcohol precipitates the albumin in the tubes and locally sterilises it. The application should be made at least once a day. In the extensive caries at times seen in the deciduous teeth, the application of nitrate of silver every two or three months and the regular use of alcohol will often arrest the caries and give the patient the use of a masticating surface —a point of the greatest importance.

(4) THE OPERATION OF CROWNING.

The operation of crowning may be resorted to where the destruction of the crown by caries is so extensive as to prevent the introduction of reliable fillings.

Teeth or roots which are to be crowned must be rendered quite free from periodontitis, and the root canals rendered sterile. The apex of the root canal should be filled before any reaming of the canal is carried out in order to prevent injury of the periodontal membrane by foreign matter.

Before crowning it is of the utmost importance that X-ray evidence should be available to see if there is any evidence of periapical rarefaction ; such rarefaction is evidence that infection has been present and it is always very difficult, if not often impossible, to be certain that sterilisation of this area has been attained. For the treatment of pulpless teeth and the difficulties attendant thereon, see Chapter XIV.

Teeth which show such areas are better extracted ; if they are not, the possibilities and conditions to which such teeth may give rise should be explained and the onus of their retention thrown on the patient. Such teeth when crowned may remain functional and give rise to no local signs or symptoms, but it must be remembered that periapical infection in a pulpless crowned or filled tooth is a form of closed sepsis which may ultimately affect the patient's health profoundly.

Collar crowns should not be used unless the collar ends well above the gingival margin and fits so accurately that it cannot give rise to stagnation areas that will injure the periodontal membrane and give ingress to toxins. The use of large gold inlays as restorations has of recent years very much lessened the necessity for crowns on the posterior teeth, and it is the custom in this country at present only to use such type of crown for the anterior teeth as ends flush with the tooth, whether of cast metal base or otherwise.

(5) THE OPERATION OF EXTRACTION.

In all cases of caries, where one of the above methods cannot be employed the tooth should be removed. For the methods of extracting teeth, see Chapter XXVII.

CHAPTER XII

Destruction of Teeth from Causes other than Caries : Abrasion, Attrition.

In caries of the teeth, the tooth substance is gradually destroyed by the action of acids and micro-organisms. Progressive destruction of the tooth substance may, however, be caused by other agencies, and of these the most important is abrasion.

(A) ABRASION.

The term **abrasion** is used to denote the destruction of the tooth substance by friction with a foreign body ; such is commonly produced by dentures, clasps and bands, clay pipes, tooth-brushes, and dentifrices, and rarely by such things as abrasive pencils sold to remove stains from teeth and files used by people endeavouring to make their artificial dentures fit more comfortably. Artificial abrasions have been practised by savages as tribal marks, but are dying out as civilisation advances (see fig. 444). Cobblers (boot lasters and clickers) by holding a ready supply of nails, thirty or so, in their mouths and serving them out with their teeth wear away the anterior teeth so as to produce a condition of anterior open bite. The action of a clasp on a denture produces a depression, the surface of which presents an eburnated appearance. Friction from the use of a clay pipe results usually in destruction of part of the occlusal portion of one tooth in one jaw and two teeth in the opposite jaw (see fig. 63).

The commonest cause of abrasion is the tooth-brush, especially when used in combination with abrasive tooth-powders. With the gradual recession of the gum, the neck of the tooth becomes exposed, and the tooth-brush, if used transversely, will tend to produce V-shaped cavities in the dentine. Such cavities are mostly found upon the maxillary canines and the mandibular canines and first premolars—the teeth which form the prominent parts of the curves of the arch. Abrasion is often more marked upon the left than the right side, and this is no doubt due to the fact that when the tooth-brush is used with the right hand—the common practice—more force is naturally applied on the left side of the mouth.

Occasionally the destruction of the enamel surface may be caused by the use of the tooth-brush and give rise to the condition usually known as 'erosion.' In this type of abrasion the appearances of the teeth vary. The labial surfaces of the anterior teeth may be affected, in which case the condition may be limited to one tooth or may involve several. The earliest indication is a slight depression which gradually deepens and extends towards the incisive margin. Gradually a cup-shaped depression is formed, which is deepest towards the cervical margin and becomes

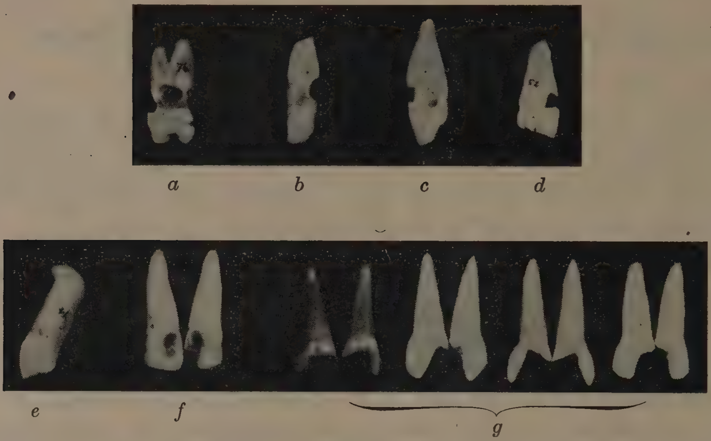

FIG. 444.—(*a*, *b*, *c*, *d*) Abrasions made by denture clasps, in the case of (*d*) the denture had not been removed from the mouth for 28 years ; (*e*) An unerupted maxillary permanent canine from an engineer aged 35 who wore full upper and lower dentures ; as the disto-occlusal portion of the tooth began to erupt palatally he filed away this portion as it interfered with the fit of his denture, and so made an artefact abrasion : the file marks can be seen on the specimen ; (*f*) two maxillary permanent first incisors, female aged 36 (*circ.*), through smoking cigarettes the teeth became stained, an abrasive pencil sold for the purpose was purchased from a chemist to remove the stains, and used to such effect that a considerable portion of each tooth was rubbed away ; (*g*) four pairs of maxillary permanent first incisors, from natives of Ilorin, Northern Nigeria, showing abrasive tribal mark. The practice is now rapidly dying out.

(London Hospital Dental Museum.) (E. S., A. Bench, W. S. Herman.)

shallower as the incisive edge is reached. Fig. 445 is an example of an advanced case. Sometimes the teeth look as if they had been pared down with a knife in a direction sloping from the labial to the lingual surfaces, and, when the mouth is closed, a considerable interval is left between the maxillary and mandibular teeth (fig. 446). To the naked eye, the teeth attacked appear hard and polished, with, occasionally, a darkish film on the surface. The dentine exposed is hypersensitive.

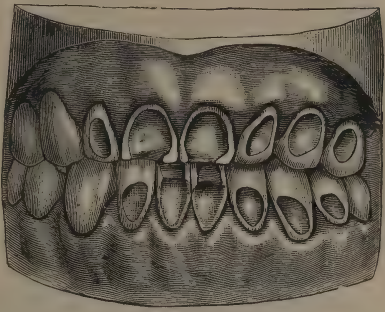

Fig. 445. (From 'The American System of Dental Surgery.')

Sections of the dentine show the fibrils or tubes ending abruptly as if cut with some sharp instrument. The tooth substance adjacent to the eroded surface cannot be stained.

Changes in the Pulp.—In teeth showing abrasion, formative activity occurs in the pulp, and results in the occlusion of the dentine tubules by calcification of their contents and the formation of secondary dentine at the pulp end of the affected dentinal fibrils (fig. 451). If the abrasive process is rapid, the dentine

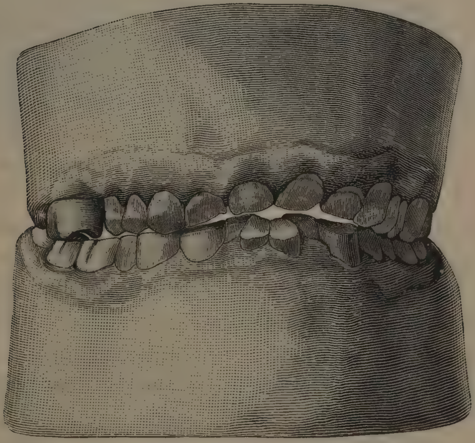

Fig. 446.

may be destroyed more quickly than the protective dentine is formed, in which case the pulp becomes exposed.

Experimental Investigation.—For many years the cause of the curious destruction of the tooth tissue termed 'erosion' was obscure. Various theories were advanced to explain the condition, but it has now been demonstrated by clinical observations and experimental evidence that 'erosion' is mainly to be attributed to the abrasive action of gritty tooth-powders and/or stiff-bristled tooth-brushes. The earliest investigations were made by Berdmore, who was the Court Dentist to George III, and he refers to his experiments in the following words :—

'I fastened in a vice a sound and well-enamelled human tooth, placing the convex side uppermost. I then took a brush, wetted and charged with a certain tooth-powder, which I had bought for the purpose, and in less than an hour, by rubbing quickly with this brush and powder, I wore away entirely the enamel of the part which was exposed to their action.

'The like experiment I repeated with all the different tooth-powders which are sold in this town, and found the same effects varied only a little in time, according to the coarseness or fineness of the powder, and the different hardness of the enamel.

'Now it is well known that a number of people brush their teeth with powders of this kind two or three times a week ; and if we allow that the brush and powder generally act on the front teeth briskly for one-fourth of a minute each time, in the space of a month they act three minutes, or in two years seventy-two minutes ; that is to say, in the space of two years the teeth have undergone a great deal more brushing than was found sufficient to destroy the finest and best enamel.

'Hence, those that brush with powders only once a week do not destroy the enamel in less than five or six years, and those who use powders but rarely can never be brought to believe that their teeth are injured by them, because the destruction creeps on too slowly to be observed.'

This work of Berdmore seems to have been forgotten. Miller more than a century afterwards published a series of articles [1] in which he conclusively showed that 'erosion' so-called was commonly due to the abrasive action of the tooth-brush and tooth-powder.

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[1] The student is advised to read W. D. Miller's articles on this subject in the *Dental Cosmos*, January, February, March, 1907.

Fig. 447 shows the result of brushing for eighteen hours with a tooth-paste which is in common use. The amount of friction applied to this tooth roughly corresponded to brushing for ten seconds daily for eighteen years. The tooth tissues and the gold filling in the first premolar are worn away equally.

An interesting experiment, undertaken by Miller, consisted in arranging a bar of wood opposite the cutting edges of the mandibular front teeth at a distance of from 2 mm. to 3 mm., and brushing for eighteen hours with an English tooth-paste. Not only were grooves produced near the necks of the teeth, but a considerable amount of tissue disappeared at the cutting edges. This experiment approximately simulates the conditions where the teeth are brought almost edge to edge when being brushed so that the bristles of the brush work freely along the cutting edges of the teeth and produce abrasion. The condition shown in fig. 446 is brought about in this way.

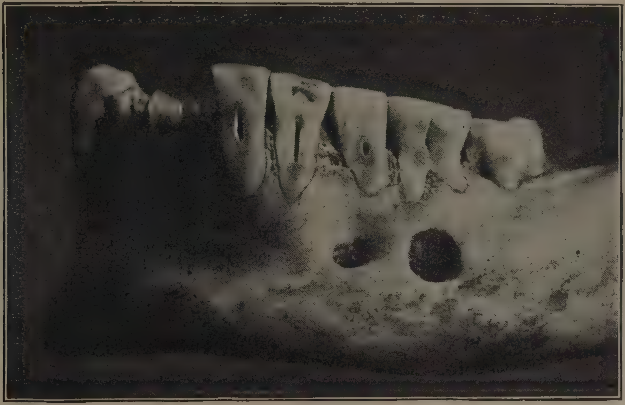

FIG. 447. (Miller.) (From *Dental Cosmos.*)

'Erosion' is considered by several writers to be due to the action of acid on the tooth tissues. The action of acids varies on the different tooth tissues. 'Some acids form soluble and others insoluble compounds which may be precipitated upon the surface of the tooth or in its superficial layers.' Acid action leaves a rough surface, in 'erosion' the surface is smooth and polished.

Workers in dynamite manufactories often suffer from a disease which causes the loss of the crowns of the mandibular incisors and the cutting edges of the maxillary incisors, the defective portions of the teeth presenting a smooth polished surface. Miller discovered that a similar effect could be produced by exposing teeth to the vapour of sulphuric and nitric acids (the acids used in the manufacture of dynamite), and then slightly brushing the affected surfaces. Here the eroding body is nitrogen peroxide.

If teeth be acted upon by a constant stream of dilute acid solution, destruction of the tooth substance will ensue, but the surface will be rough, showing acid action, and not smooth as in cases of abrasion.

G. V. Black [1] experimented as follows: The roots of two fresh healthy premolars were enveloped with gutta-percha so that only the crowns were exposed; the teeth, with their approximal surfaces together, were then placed in a jar containing dilute hydrochloric acid (1 in 400) which, by means of an ingenious apparatus, was set in motion; the teeth were arranged in the jar in such a position that a current fell upon their outer surfaces but struck one of the surfaces with greater force than the other. The result was that the cusps disappeared and a groove was formed between the teeth, this groove being more marked upon the surface which received the greater force of the current. Many other experiments were made, and it was found that strong solutions produced general softening, while a solution of 1 of acid in 5,000 of water had no appreciable effect after three months' trial.

Head [2] has investigated the action of acids on enamel, and has shown that the structure resists the decalcification of acids and acid salts most unevenly and erratically. He has also shown that the saliva considerably inhibits the action of carbonic acid and many other acids, such as lactic, citric, etc.

The result of an experiment undertaken by Miller to show the difference between mechanical friction alone and mechanical friction plus acid action is shown in figs. 448 and 449. The teeth in the left half of the mandible were treated with a $\frac{1}{2}$ per cent. solution of acid calcium phosphate applied on cotton-wool [3] and in the right half with water, also applied on cotton-wool.

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[1] 'American System of Dental Surgery,' i, 1004.

[2] *Dental Cosmos*, xlix, 801.

[3] Miller in this experiment does not say how long the acid calcium phosphate was allowed to remain in contact with the tooth.

Both sides were brushed twice a day with different powders for two and one-third years. The destruction is much more marked on the left side, which was treated with acid calcium phosphate.

Etiology.—Experimental evidence suggests that 'erosion' is really a type of abrasion and due to the mechanical action of the tooth-brush and gritty powder. The question arises: Is it possible that acid agents may be present in the mouth in certain pathological conditions and so aid the abrading process? Many authors maintain that 'erosion'[1] is intimately associated with 'gout,' and that in gouty subjects acids capable of affecting the tooth tissues are produced in the mouth. Clinical observations on patients in private practice certainly tend to show that waste of tooth tissue is more marked in those with a gouty diathesis than in those without. On the other hand, Miller and S. P. Mummery examined patients in the hospitals of London and Berlin and found no case of erosion in twenty-four cases of gout and ninety-nine of rheumatism (eighty-nine chronic and ten acute). Caries at the necks of the teeth was found in many of the patients, but no disintegration of the enamel. Riegner, of Breslau, also examined amongst hospital patients two cases of gout and 115 cases of articular rheumatism and found no wasting of the teeth. These data are interesting in view of the fact that hospital patients rarely use a tooth-brush.

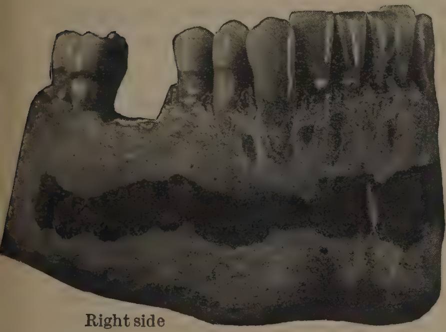

FIG. 448. (Miller.)

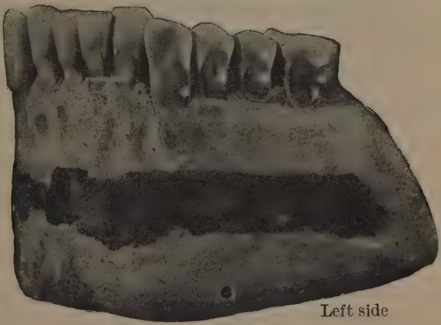

FIG. 449. (Miller.)

(From *Dental Cosmos.*)

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[1] The term 'erosion' has been allowed to remain in the text in several places as it is difficult, and perhaps inexpedient, to do away altogether with a term which has been so universally used for many years.

Brubaker[1] considers that the source of the acid which affects the teeth is to be found in an abnormal secretion of the mucous glands of the cheeks and lips. These glands are muciparous, and their secretion is composed of water, mucin, and inorganic salts, the chief of which, sodium phosphate, imparts to the fluid its alkalinity. In certain conditions, such as 'gout,' the secretion increases in quantity and is acid in reaction, the acid sodium phosphate which is formed acting as the decalcifying agent.

Kirk,[2] who has carefully studied the question of the connection between the saliva and erosion, is of opinion that 'erosion' is intimately connected with the gouty and rheumatic diathesis.

He considers that when erosion is 'general,' that is, when it attacks the teeth generally, lactic acid is the solvent, but that, when the condition is localised, the agent is acid sodium phosphate or acid calcium phosphate derived from abnormal secretion of the mucous glands.

Cook[3] contends that mucic acid is an important acid agent in the production of 'erosion,' and thinks that it is produced by a series of changes from lactose. It is, however, an open question whether mucic acid is ever present in the mouth.

Michaels[4] states that, where 'erosion' is found, the saliva of the patients contains an increased amount of sulphocyanide in the form of the ammonium and potassium salts and that by submitting the tooth to a slow drip of a solution of sulphocyanide of potassium for a few days he was able to produce conditions similar to erosion.

A marked destruction of tooth tissue is occasionally seen in patients who consume a large amount of fruit in the form of oranges, grapes, and lemons, but in these cases the effect produced on the teeth is not the same as in cases of 'erosion.' In the case of a patient who was frequently sucking lemons and had acquired the habit of sweeping the tongue over the labial and then over the palatal aspects of the maxillary teeth it was found that the enamel had almost disappeared.

During recent years the number of patients with 'erosion' of the teeth has progressively diminished and the condition is now relatively seldom seen. Concurrently, the materials used for cleaning the teeth have undergone a change, tooth-pastes having largely superseded tooth-powders, and people are now better instructed as to how to brush their teeth and what type of brush to use. The friction agent has therefore been disappearing from the dental toilet, but conditions likely to give rise to abnormal secretion of the salivary glands have still been present.

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[1] *Internat. Dent. Journ.*, 1894, 742.

[2] *Items of Interest*, July 1902, and *Dental Review*, May 1903.

[3] *Dental Review*, May 1906, 461.

[4] *Internat. Dent. Journ.*, xxi, 248.

From the foregoing statements it will be seen that varying opinions as to the cause of ‘ erosion ’ are held by those who have investigated the subject. The facts elicited from a general review of all the information at present at our disposal would seem to show conclusively that **the condition is nearly always due to the abrasive action of the tooth-brush and tooth-powders.** Occasionally, however, no etiological factor can be found—for instance, when this non-carious loss of tooth substance occurs on the buccal aspect of the molars of Australian aboriginals.[1]

Treatment.—In the first place, tooth-powders should be avoided and a soap with a soft brush used for cleansing purposes. The teeth must on no account be brushed ‘ crosswise.’ If the abraded areas are painful, they may be touched with phosphoric acid, chloride of zinc, or, if in a position not exposed to view, nitrate of silver may be used, or a paint may be prescribed, the patient being instructed to dry the tooth and paint it with the solution, keeping the tooth dry to allow the material to harden. The following will be found useful :—

℞	Gum-mastic (powder)	ʒi
	Zinc chloride	gr. v.
	Chloroform	℥ss

Mix. To be used as a paint.

If the cavities are deep and are likely to harbour food debris, they should be filled.

(B) ATTRITION.

Attrition is a wearing away of the tooth substance, caused by the grinding of one tooth surface over another. It is seen mostly in the deciduous teeth and in the teeth of the aged. The amount of attrition depends to a great extent upon the character of the food, the density of the tooth substance, and the power of mastication. The surfaces of all the teeth may be affected, or only one or two teeth may be attacked. When confined to one or two teeth, the attrition is generally due to some irregularity of the bite. Fig. 450 shows a good example of attrition due to an irregularity of the bite—the mesial angles of the maxillary first incisors impinge upon the mandibular first incisors.

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[1] ‘ Dentition and Palate of the Australian Aboriginal,’ Campbell, T. D., 1925. Plate XLIV, figs. 1 and 2.

Broca has suggested the recognition of **four degrees of wear** :—

In the **first,** the enamel alone is worn without any obliteration of the cusps ; in the **second,** the tubercles of the crown have disappeared, and the dentine is exposed ; in the **third,** the height of the tooth is still further reduced ; in the **fourth,** the wear has extended to the neck, the crown having entirely disappeared, and either the pulp cavities are invaded or barriers of secondary dentine have formed.

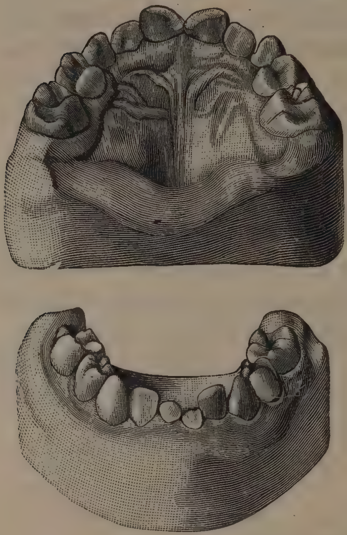

Fig. 450.

Involuntary grinding of the teeth, leading to undue attrition, is not uncommon in children and is sometimes met with in adults, more especially those with gouty or rheumatic tendencies. Marie and Pietkiewicz[1] state that the habit of grinding the teeth frequently develops in the course of disorders of the central nervous system.

Examined microscopically, the dentinal tubes in cases of attrition will be seen to end abruptly at the worn surface, their lumen is occluded by calcification of their contents, so that the dentine tubes radiating to the worn surface form a translucent zone impermeable to stains from either end. The pulp chamber will show the formation of secondary dentine, opposite the ends of the affected tubes, but the formation of secondary dentine will not always be found to have kept pace with the destructive process.

Treatment.—In most cases treatment is unnecessary. If the posterior teeth have been lost, and the anterior teeth are being worn away through bearing the brunt of mastication, the

[1] *Revue de Stomatologie,* March 1907.

bite should be taken off the anterior teeth by means of dentures. If the patient refuses to submit to dentures, the progress of the destruction can be arrested by filling the cavities with metal and bringing the filling over the edges of the enamel in such a way that, in occlusion, the fillings come in contact and so arrest the progress of tooth destruction. The latter treatment is effectual, but the appearance produced is somewhat unsightly.

The hypersensitiveness of dentine, which at times accompanies rapid attrition, should be treated as indicated on p. 392.

CHAPTER XIII

Diseases of the Pulp.

The Protective Action of the Pulp—Local Reaction to Injury (Inflammation)—Regressive Changes—Fibrosis—Fatty Degeneration—Colloid Degeneration—Calcareous Deposits.

(A) THE PROTECTIVE ACTION OF THE PULP.

WHENEVER the dentine is injured a definite response takes place in the dentine fibrils which have been exposed to injury and, later, in that portion of pulp contiguous to their pulpal terminations.

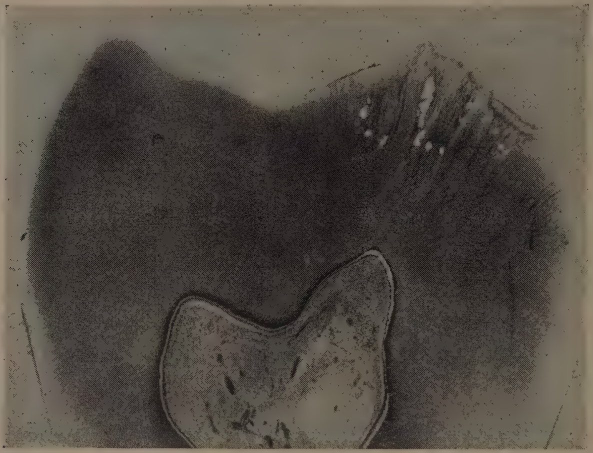

FIG. 450A.—An early response of the tooth pulp against the advance of caries; being a decalcified section, the translucent zone in the dentine is not seen. (E. S.)

The response is governed by the vitality of the pulp and is evidenced by the calcific occlusion of the tubes in the injured dentine and the production of a modified type of dentine known as secondary dentine. If this response of the pulp did not occur, the pulp cavity would be more frequently exposed and the life of the pulp jeopardised.

Hunter was the first to recognise this property of the pulp, for he remarks that: 'A tooth often wears down so low, that its cavity would be exposed if no other alteration were produced in it. To prevent this, nature has taken care that the bottom part of the cavity should be filled up with new matter, in proportion as the surface of the tooth is worn down.'

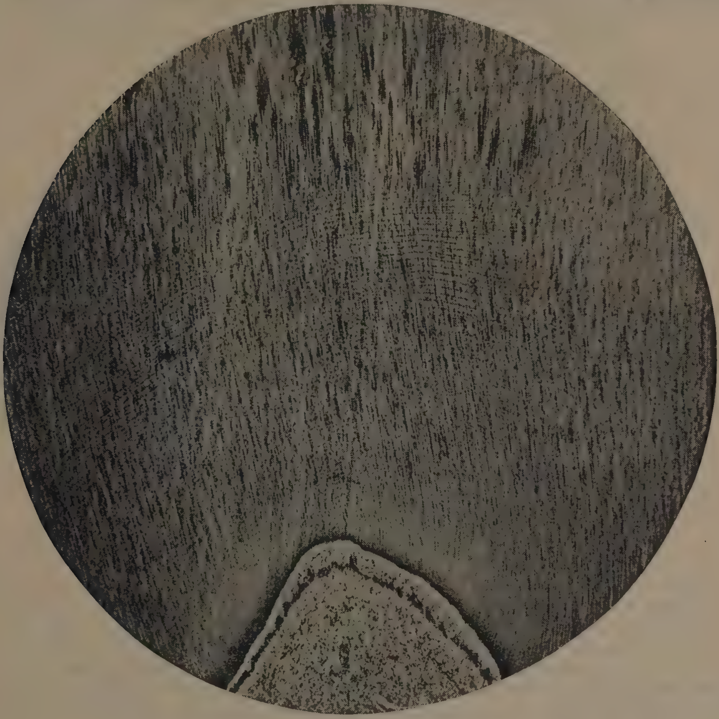

Fig. 450b.—Higher magnification of the affected cornu of pulp shown in Fig. 450a. (E. S.)

Salter[1] devoted considerable attention to this phenomenon and suggested, for the new tissue, the term 'dentine of repair'; while Hopewell-Smith has named it 'adventitious dentine.'

The new formation may be observed in its simplest form in

[1] 'Dental Pathology and Surgery,' 1874.

cases of abrasion and attrition where, the injury to the dentine being of a slow progressive nature, the pulp is not hurried in its task and the freshly formed tissue is strictly limited to the area of the affected fibrils, as shown in fig. 451.

It will be observed that in the affected area the fibrils have become calcified, a fact which shows that reaction also takes place there. The calcification of the affected fibrils is shown by the fact that stains will not penetrate the dentine tubes which they occupied from either end.

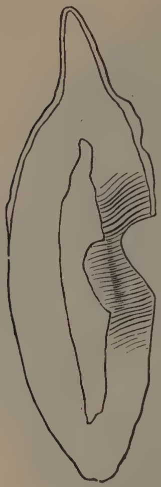

Fig. 451.—Section through a tooth affected by abrasion; the formation of secondary dentine is strictly limited to the pulp region of the injured fibrils which are occluded by calcific deposit.

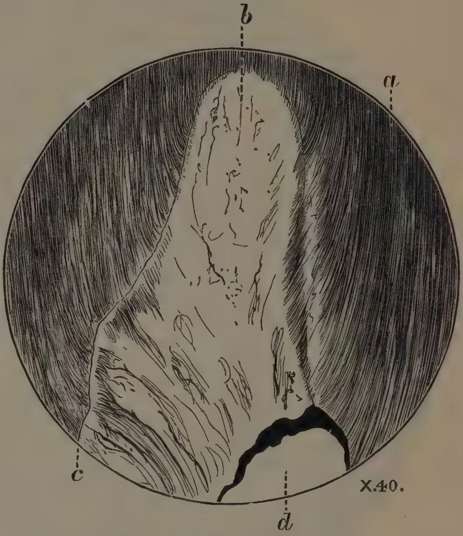

Fig. 452.—Section through a tooth the dentine of which was exposed by attrition: (*a*) primary dentine; (*b*) secondary dentine; (*c*) line of junction; (*d*) pulp chamber.

There is usually a definite line of junction between the primary and secondary dentine; this is shown in fig. 452.

The structural perfection of secondary dentine, *i.e.* its likeness to the primary dentine, depends on the age when it is formed, the functional health of the tooth pulp, and the rapidity with which it has to be produced; and this last will depend on the nature of the stimulus or injury necessitating its production.

The structure of secondary dentine varies considerably in different circumstances. Hopewell-Smith has described[1] five varieties—*fibrillar*, *areolar*, *laminar*, *cellular*, and *hyaline*.

In the **fibrillar** form the tissue may approximate to normal fine-tubed dentine, an abrupt bend of the tubes being all that there is to mark the junction of the two tissues. In many specimens, however, the tubes are less regular and not so plentiful as in fig. 452. The fibrillar variety of secondary dentine follows prolonged injury of a low intensity, such as attrition.

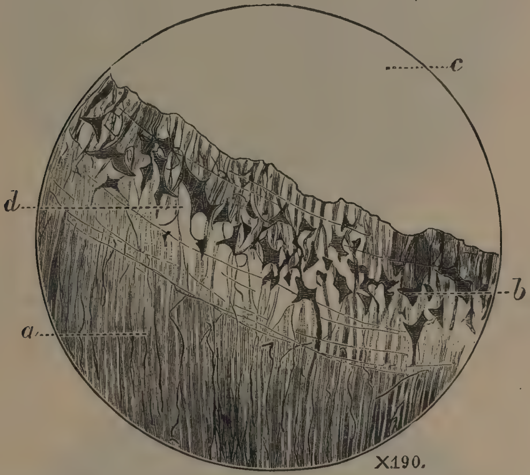

Fig. 453.—Areolar secondary dentine. (*a*) carious dentine; (*b*) interglobular spaces; (*c*) pulp cavity; (*d*) newly formed and irregular-tubed dentine.

The **areolar** variety (fig. 453), which in its general characteristics resembles a dentine filled with interglobular spaces, would seem to occur when the new tissue has been formed rapidly as the result of rapid caries. Such newly formed tissue possesses but little resistance to the carious process and becomes quickly infected (figs. 454 and 455).

The **cellular** type differs from the areolar variety in containing, in the matrix, cells which may be fusiform or round (fig. 456).

The **hyaline** variety is a clear, homogeneous, structureless deposit of dentine (fig. 457); and the **laminar,** as the name suggests, is tissue with a laminated form (fig. 458).

Clinical observations show that the reparative power of the

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[1] See *Trans. Third International Dental Congress*, Paris, 1900.

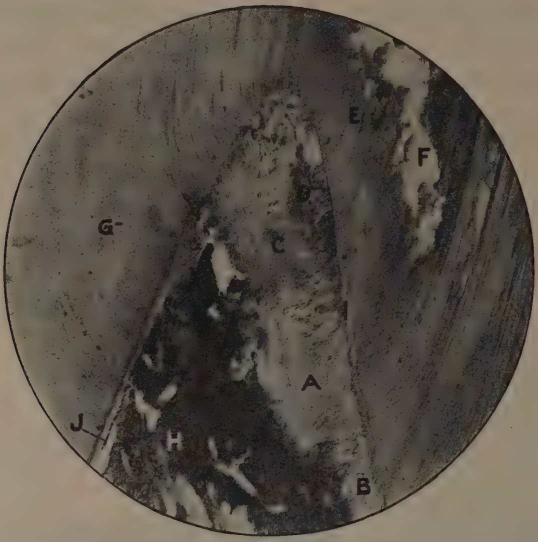
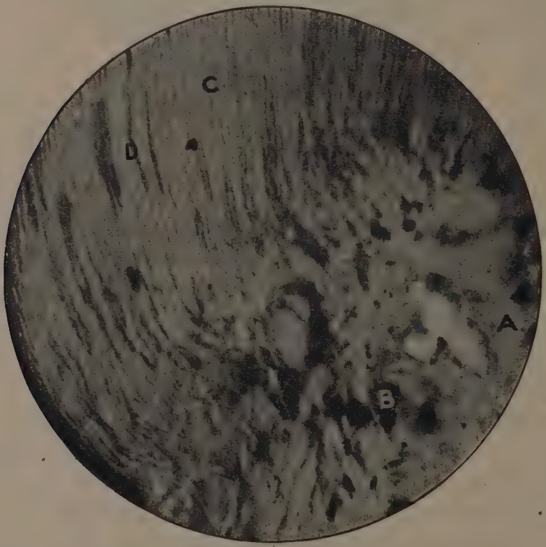
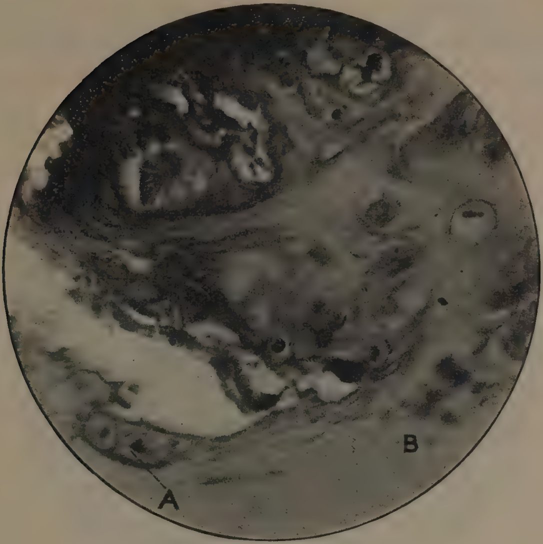


FIG. 454. Areolar and fibrillar secondary dentine. (A) secondary dentine with fibrillar structure at B, and areola at C, which at D is infected with micro-organisms from caries at E; (F) carious cavity; (G) primary dentine; (H) pulp tissue showing signs of reaction; (I) junction of primary and secondary dentine; (J) layer of odontoblasts. (A. Hopewell-Smith.)



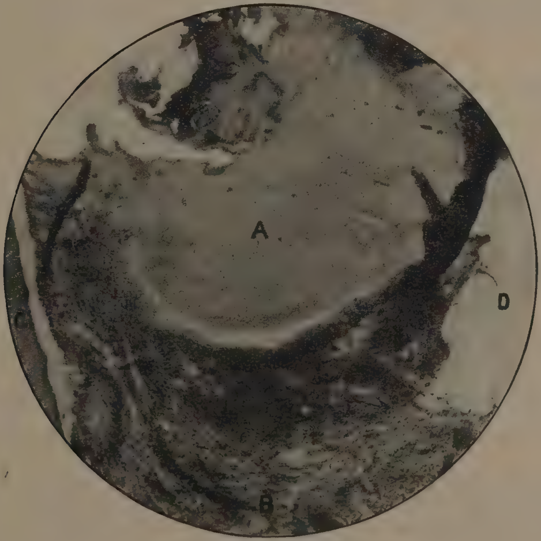
× 300.

FIG. 455. Secondary dentine infected by micro-organisms from the primary dentine. (A) secondary dentine; (B) masses of micro-organisms; (C) primary dentine; (D) carious dentine. (A. Hopewell-Smith.)



× 250.

FIG. 456. Cellular secondary dentine. (A) encapsuled cells with nuclei; (B) structureless matrix. (A. Hopewell-Smith.)



× 40.

FIG. 457. Hyaline secondary dentine (A) ground-glass-like dentine; (B) pulp tissue showing reaction; (C) primary dentine; (D) abscess cavity. (A. Hopewell-Smith.)

pulp varies considerably in different individuals ; in some cases, the tissue destruction by caries is well balanced by the formation of new tissue, while, in others, the power of repair seems to be almost entirely lacking.

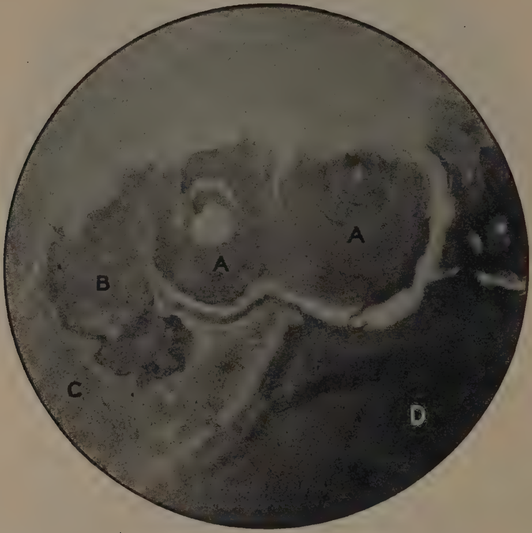


FIG. 458. Laminar secondary dentine. (A) laminar dentine ; (B) cellular dentine ; (C) pulp tissue showing signs of reaction ; (D) primary dentine. (A. Hopewell-Smith.)

(B) REACTION TO INJURY—INFLAMMATION.[1]

The reactions of the tooth pulp to injury result in inflammation. Two grades of inflammation are recognised, namely, acute and chronic ; in the former, the development of the inflammatory process is rapid and is accompanied by marked clinical symptoms, while, in the latter, the reaction is slow in development and is accompanied by feebly marked clinical signs —indeed, in some instances, no clinical signs are apparent.

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[1] Colloquially, and unfortunately customarily, inflammation of the pulp is often known by the etymologically unsound name of 'pulpitis' ; the pulp of a tooth is in many ways analogous to the marrow of bone ; it would be in keeping with surgical nomenclature and more euphonious if the analogy were carried further and the term dental myelitis or odonto-myelitis used to describe inflammation of the tooth pulp just as osteo-myelitis is used to describe inflammation of the bone marrow.

(1) Acute Inflammation of the Pulp.

Causes.—Acute inflammatory reaction in the pulp may arise from *trauma*, such as rupture of the apical vessels from dislocation of the tooth, fracture of the tooth involving the pulp, or laceration of the pulp tissue in the preparation of the tooth for filling. Another group of causes is the injudicious use of *physical and chemical agents*—for example, the freezing of teeth with ethyl chloride, or the use of a drug like arsenic for the treatment of sensitive dentine. The commonest cause of acute inflammation of the pulp is injury from *bacterial toxins* which react the pulp tissue either through a carious cavity, or, more rarely, via the periodontal membrane, as in cases of advanced parodontal disease.

Morbid Anatomy and Pathology.—The changes in the pulp tissue resulting from injury are similar to those which occur in other vascular structures. The process may terminate in *resolution* when the injury has taken the form of a concussion or slight dislocation of the tooth. Resolution may follow after injury of the apical vessels, but this is rare, and in this connection the following case recorded by A. S. Underwood [1] is interesting. Whilst performing immediate torsion on a maxillary second incisor, the tooth was completely dislocated and fell on the floor. It was sterilised and replaced. Two years afterwards the tooth responded to thermal changes and showed no degenerative changes (see also p. 889).

In other cases the reaction in the tissues may be so intense in character as to lead to *gangrene* of the pulp. Injury to the pulp is frequently followed by gangrene, the reason being that this delicate tissue is enclosed by unyielding walls which do not allow for inflammatory œdema, so that the increased pressure in the cavity leads to vascular obstruction.

When the injury is due to bacterial infection, *suppuration* usually follows, and, where the infection is intense, ‘ gangrene ’ may occur. When suppuration is once established in a part of the pulp, the remainder rapidly becomes involved and the whole of the tissue is eventually destroyed.

Stages of suppuration of the pulp are shown in fig. 459. In the zone (*f*) bordering on the abscess, the cells show a tendency to the formation of fibroblasts, and this would seem to indicate protective action on the part of the pulp to cut off the pus from

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[1] *Trans. Odonto. Soc.*, xviii, 98.

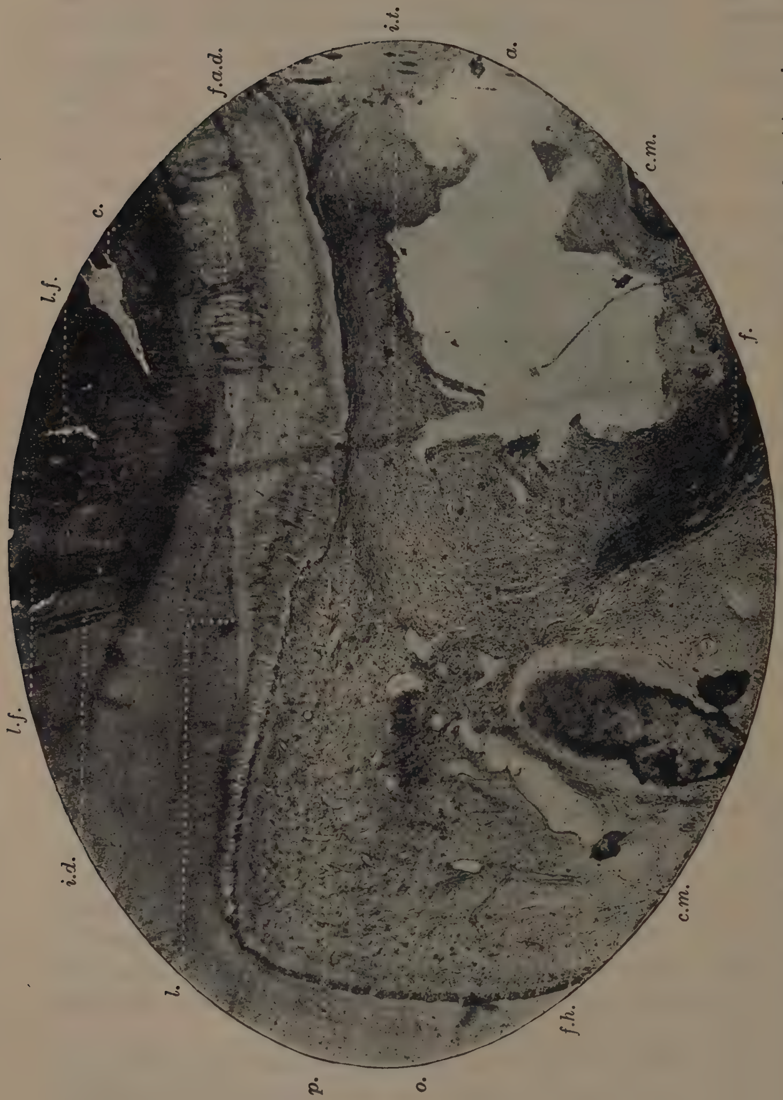

FIG. 459.—Vertical section of human molar tooth affected with caries and inflammation of the pulp. (*c*) carious cavity; (*i.d.*) dentinal tubes infected with micro-organisms; (*l.f.*) liquefaction foci; (*l.*) original limit of the pulp cavity; (*f.a.d.*) fibrillar secondary dentine; (*p*) pulp tissue apparently but slightly affected by the inflammatory process; (*o.*) original odontoblasts; (*i.t.*) acute inflammation of the pulp tissue; (*a.*) abscess cavity; (*f.h.*) hyperæmic blood-vessel; (*c.m.*) deposit of calcoglobulin; (*f.*) fibroblasts.

(From 'The Histology and Patho-histology of the Teeth.' A. Hopewell-Smith.)

the remainder of the tissue. In the tusks of elephants examples of the encapsulation of pus are often met with, and in man occasionally this protective action of the pulp is able to localise the infection so that a small chronic abscess is confined to one cornu of the pulp while the remainder appears almost normal (fig. 460). Gysi[1] records a case where an abscess in the cornua of a maxillary first molar had been isolated from the rest of the pulp by a wall of secondary dentine, and Miller[2] figures a tooth in which the appearances suggest that an abscess cavity had been bridged over and separated from the main pulp cavity.

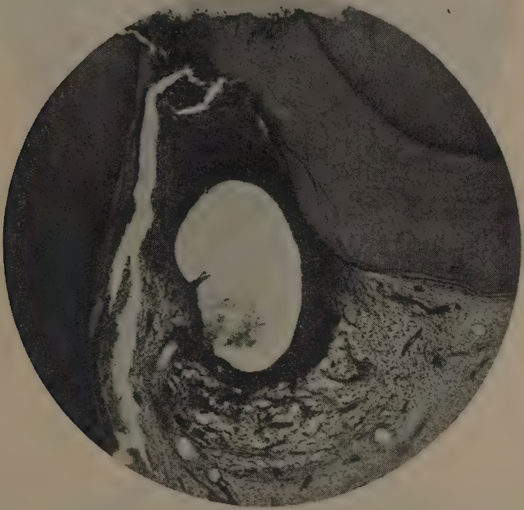

Fig. 460.—After 48 hours' severe pulp pain the removal of carious dentine gave egress to a bead of pus. (G. B. Pritchard.)

Occasionally the pulp exhibits a remarkable power of recovery from injury, as shown in cases where an unsuccessful attempt has been made to remove a tooth and a portion of the root containing a living pulp has been left behind, the superficial portion of the pulp in time being replaced by secondary dentine. A detailed account of such a case is given by C. S. Tomes.[3] Sections of this tooth showed that the widely exposed pulp had been covered in by a layer of secondary dentine, and that, in places, fragments of the original dentine had become embedded in the new tissue (see figs. 461 to 463).

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[1] *Schweizerische Vierteljahrsschrift für Zahnheilkunde*, 1900, 254.

[2] *Dental Cosmos*, xliii, 850.

[3] *Trans. Odonto. Soc.*, xxviii, 183.

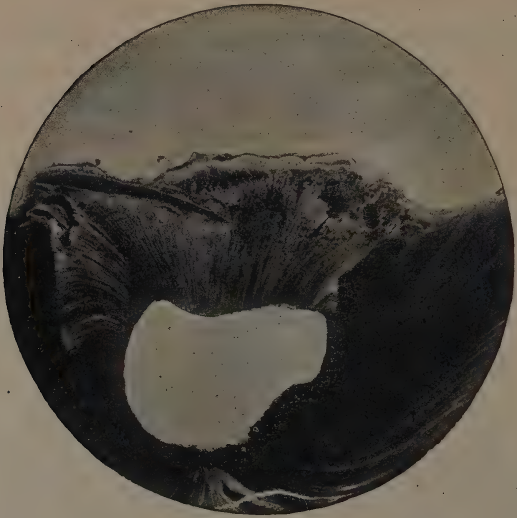

Fig. 461.—General view of the secondary dentine, showing the pulp cavity beneath it and the dentine of the original tooth, which appears very dark. A thin strip broken off the old dentine is embedded in the secondary dentine. (From *Trans. Odonto. Soc.*)

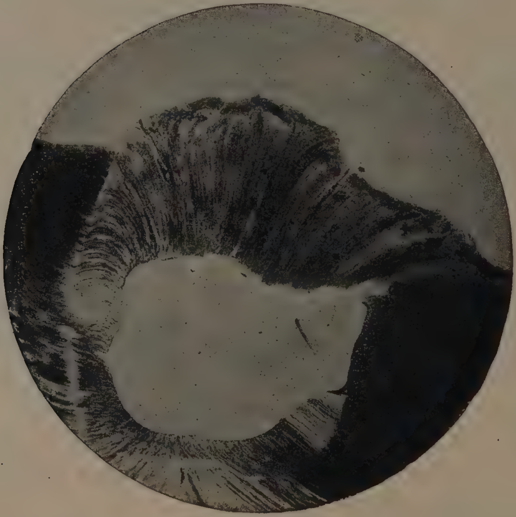

Fig. 462.—Another view of the secondary dentine. (From *Trans. Odonto. Soc.*)

A comparable condition is shown in fig. 464, which is a section of a mandibular left permanent first molar from a boy aged 15. The crown had been broken in an attempt at extraction, leaving the pulp exposed and the lingual aspect of the tooth fractured well below the gum margin. The tooth pulp did not die but became joined to the gum on the lingual aspect and the whole covered with epithelium ; several splintered fragments of dentine had become incorporated in the exposed pulp and had not only become completely covered with soft tissue but had new calcified tissue formed around them. On two of the fractured surfaces new calcified tissue has been laid down, apparently in direct continuity in places.

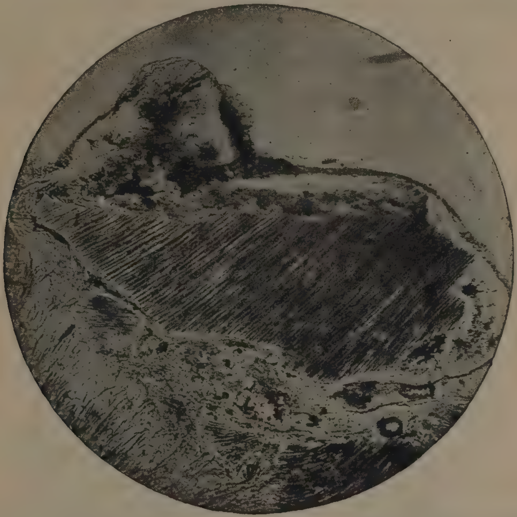

FIG. 463.—A portion of the displaced original dentine embedded in the secondary dentine. (From *Trans. Odonto. Soc.*)

Bacteriology.—Cultural examinations of acutely inflamed pulps show the presence of streptococci, staphylococci, and diphtheroids.

In twelve consecutive cases examined the following organisms were present :—

Streptococcus pyogenes	4 cases
,, *brevis*	8 ,,
Staphylococcus albus	6 ,,
,, *aureus*	1 case
Gram diplococci	2 cases
Diphtheroids.	6 ,,

Symptoms.—The symptoms of acute inflammation of the pulp are pathognomonic—sharp shooting pain, often of a throbbing character, and generally more severe at night. Pain at night is not due to the assumption of the horizontal position with its possibly increased blood pressure in the teeth, but to the peripheral capillary dilatation which takes place as sleep comes on ; patients can usually lie down comfortably but they cannot sleep, as the pain comes on in the act of falling to sleep even when sitting up. Thermal changes also lead to severe paroxysms of pain, although in the early stages of acute inflammation cold produces relief by constricting the blood-vessels. It must be distinguished from acute periodontitis, and the main points of difference are briefly as follows :—

ACUTE INFLAMMATION OF THE PULP.	ACUTE PERIODONTITIS.
Pain sharp, throbbing, or lancinating — intermittent and reflected.	Pain dull, heavy, and constant.
Thermal changes to the teeth cause pain.	Thermal changes do not cause pain.
Pressure or tapping on the tooth causes no pain.	Pressure or tapping on the tooth causes pain.
Slight pressure on a piece of cotton-wool inserted in the cavity generally causes acute pain.	Slight pressure on a piece of cotton-wool inserted in the cavity does not cause pain, except through pressure transmitted to the periodontal membrane.

Attention to these points will assist in diagnosis, but it must not be forgotten that with acute inflammation of the pulp there is at times inflammation of the periodontal membrane through continuity of the tissues.

Treatment.—When the inflammation is due to trauma, or to the action of physical and chemical agents, there is reasonable probability that resolution will occur. Therapeutic measures must be directed towards the avoidance of fresh injury to the tooth, and counter-irritation should be applied to the gum covering the root in the form of drugs, such as strong tincture of iodine or a capsicum plaster. If the dentine has been exposed by the trauma, the exposed surface should be treated with a strong solution of nitrate of silver and, when feasible, protected from thermal stimuli by a covering of some non-irritant cement. Should the inflammation be the result of drugs used for treating sensitive dentine, the use of the drugs must be discontinued and the cavity syringed and filled with a non-conducting and non-irritant filling, such as zinc oxide and eugenol. Where, however, the clinical features suggest that resolution is not likely to occur, the pulp should be immediately removed. If a pulp is infected, or injured by bacterial toxins, experience teaches that extirpation is the best treatment, as acute inflammations of this type practically never resolve.

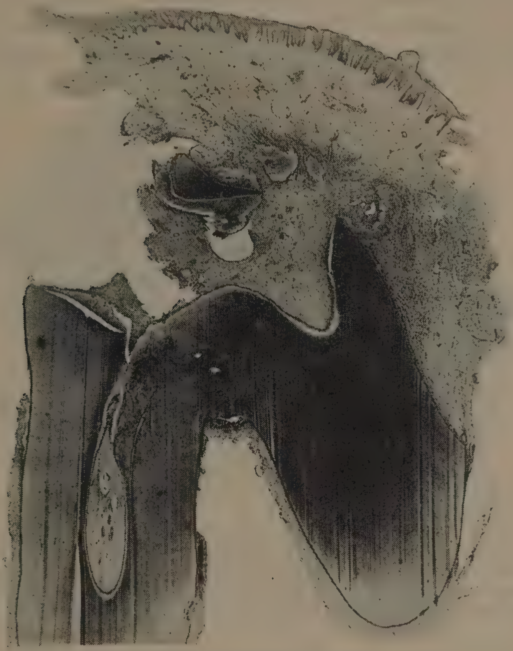

Fig. 464. From male aged 15 years. (E. S.)

(2) Chronic Inflammation of the Pulp.

Chronic inflammation of the pulp follows on injuries similar to those leading to acute inflammation. The reaction of the tissues in chronic inflammation of the pulp is different from that seen in the acute form, and this is due to the fact that in the chronic form the injury is constant, though of less intensity.

Morbid Anatomy and Pathology.—The character of the reaction of the pulp tissue, when the injury is of long duration, varies considerably. We will first deal with the condition where the pulp cavity is exposed and the pulp tissue is injured by constant doses of bacterial toxins. Suppuration usually occurs and the pulp tissue is destroyed by a progressive ulceration.

Chronic Inflammatory Hyperplasia.—In some cases cell proliferation is prominent; there may be an exuberant mass of granulations produced on the exposed surface which, as chronic inflammation always tends to bring about fibrosis, results in the formation of a mass of fine fibrous connective tissue and in time produces a flesh-like mass in the carious cavity—this condition of inflammatory hyperplasia is commonly known as a 'polypus' of the pulp. A section through such a hyperplasic pulp will show granulation tissue having a tendency to develop into fibrous tissue; in some cases there is a dense infiltration with inflammatory cells and in cases of long standing the condition of fibrosis is marked. The surface is often covered with a layer or several layers of squamous epithelium (figs. 465 A and B). Several explanations are given to account for the presence of this epithelium. Hopewell-Smith describes how the epithelium on the gum, irritated by the sharp edge of an approximal cavity, proliferates and extends by direct continuous growth on to the granulation-covered surface

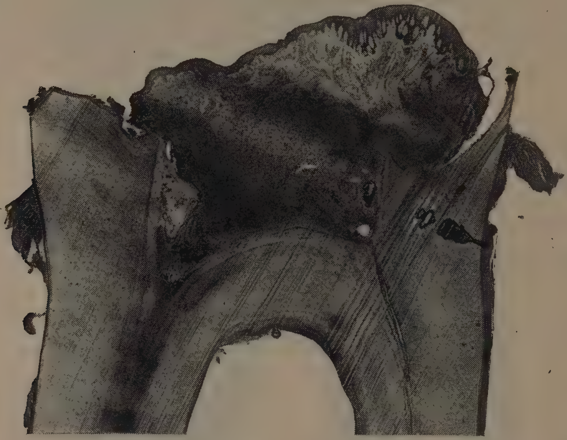

FIG. 465A.—Hyperplasia ('polypus') of the pulp, showing a fibrosed mass of granulation tissue irregularly covered with epithelium. It not uncommonly happens, as in this case, that a layer of calcific tissue is laid down where the soft tissue comes into contact with the carious surface of dentine. (E. S.)

of the exposed pulp ; no doubt this is true of some cases, but one not infrequently finds epithelium present when, owing to the isolated position of the mass, it seems impossible that this should have happened, and it seems probable that in these cases epithelial cells while still viable may have become detached from the oral lining during the act of mastication, and come to rest on the granulating surface and grown there ; and indeed a granulating surface is the ideal surface for such growth.

A granulation, reduced to its simplest terms, consists of a fibroblast and a capillary loop ; it contains no nerves, and neither does its ultimate fibrosed product in this condition of the tooth pulp, and these fibrosed masses are insensitive to pressure unless it be conveyed through it to nerves which still exist in the original pulp tissue.

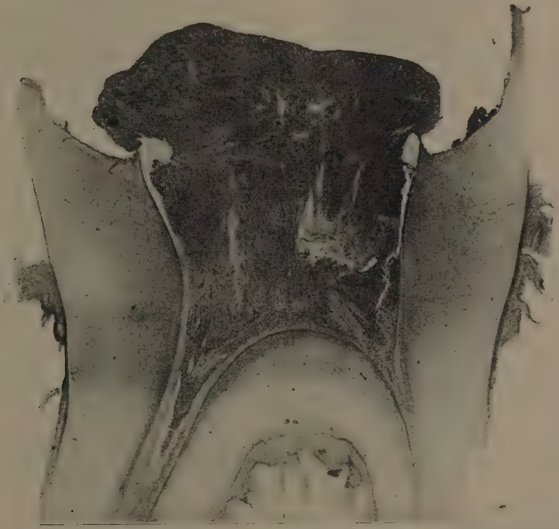

FIG. 465B.—Chronic inflammatory hyperplasia of the pulp with no epithelial covering. (E. S.)

Till 1927 such inflammatory hyperplasia had only been described in connection with the permanent teeth ; specimens were then[1] shown of the condition in the deciduous teeth. Occasionally these masses are found in direct continuity with somewhat similar inflammatory hyperplasias having origin from the gum margin or periodontal membrane (figs. 464 and 466), but such a condition is rare.

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), 1927, xx, 67. (E. S.)

Pink Spot.—There is a type of case in which the dentine and enamel are destroyed from within and the pulp tissue extrudes. The condition is more common in the incisor region than elsewhere, but it is found in other teeth, including the mandibular third molar, and its presence is first noticed by the pink appearance of the pulp through the attenuated enamel, an appearance which led to the affection being known as 'pink spots.' J. H. Mummery has published[1] a detailed account of a case and has suggested that the destruction of the dentine may be due to a chronic inflammation of the pulp. In the case he records the normal pulp tissue was replaced by granulation tissue composed of cells and connective tissue in varying proportions, and the wall of the pulp cavity showed absorption areas which, in places, had been filled in with a deposit of osseous tissue with lacunæ, but few canaliculi were apparent (fig. 467). There was a plentiful supply of blood-vessels. In places strong bundles of connective tissue fibres were attached to the walls of the pulp cavity. The cellular element of the growth consisted of endothelial cells, connective tissue cells, leucocytes, and large giant cells. The portion of the pulp in the lower end of the root was fairly normal in appearance.

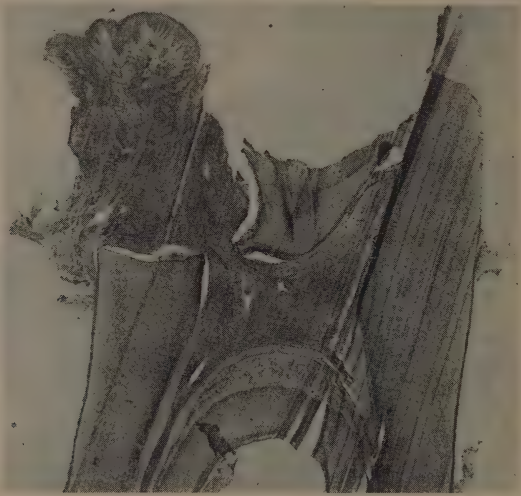

FIG. 466.—Conjoined 'polypi' of gum and pulp. Mandibular left permanent first molar. Male aged fifteen. (E. S.)

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[1] *Brit. Dent. Journ.*, 1920, April 1, 301.

Another case was examined in detail by G. B. Pritchard.[1] The tooth was a mandibular second premolar and had been watched for fifteen months before extraction of the tooth. Serial sections showed that the condition was one of absorption from within the pulp cavity ; some of the areas produced by the absorption were occupied by granulation and others by bony tissue. Pritchard regards the condition as a granuloma of the tooth pulp, and comparable to the hyperplasia (' polypus ') occasionally seen when the pulp has been exposed by caries (see p. 422). He regards both as being the result of irritation, the irritant in one being toxic and in the other possibly traumatic.

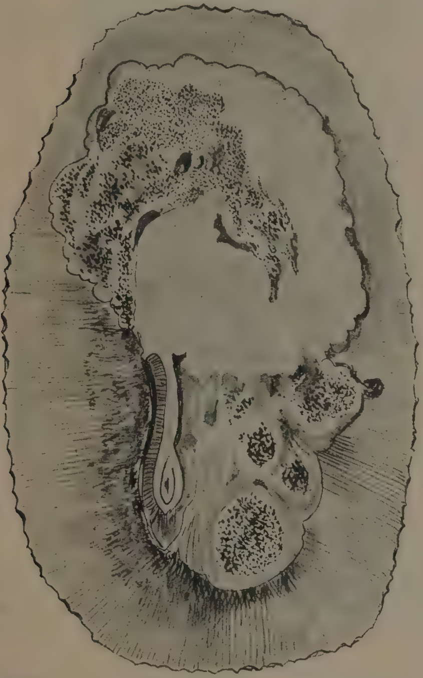

FIG. 467.—Transverse section of a mandibular incisor from a case of ' Pink Spot.' (From *Brit. Dent. Journ.*)

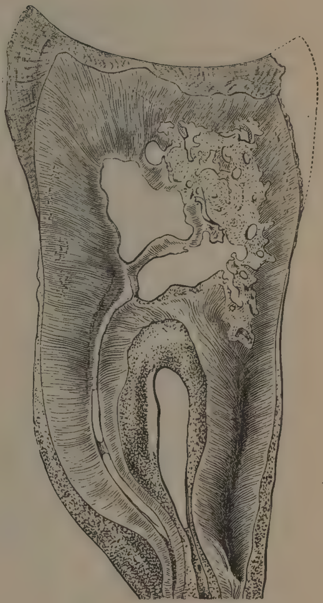

FIG. 468. (Salter.)

A case recorded by Salter [2] would seem to belong to this group. The tooth was a mandibular molar and showed three openings just below the edge of the enamel. These canals were short and horizontal, and connected with the interior of the tooth. Sections through the tooth showed that a considerable portion of the dentine had been absorbed and replaced by bone (fig. 468).

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), 1931, September.

[2] ' Dental Pathology and Surgery,' 1874, 79.

We have no definite knowledge of the cause producing the inflammation in these cases. The condition is sometimes symmetrical, a fact which suggests that a variation in development may be an etiological factor.

Lepkowski[1] has shown that in rare instances anastomoses exist in the embryo between the pulp vessels and the vessels external to the tooth germ, and it seems likely that these anastomoses may occasionally persist and give rise to the abnormality shown in fig. 48.

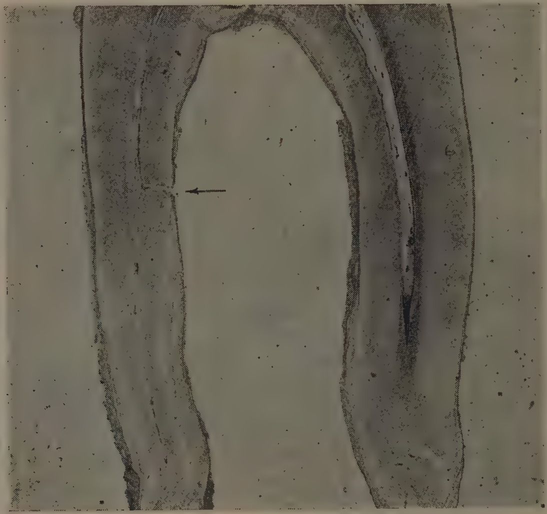

FIG. 469.—Aberrant opening into pulp canal of mandibular permanent first molar from periodontal membrane. (E. S.)

Occasionally a channel exists between the pulp and the periodontal membrane of a tooth which is not of developmental origin. Examples are shown in figs. 469, 470, 471 and 545.

It seems possible that in cases of ‘ pink spots ’ the pulp tissue may become infected via an abnormal canal. The following case supports such a view. The tooth affected was the maxillary left first incisor ; there was no parodontal disease, but the gingival margin had been destroyed in places by too vigorous use of the tooth-brush. On the mesio-labial aspect of the root about one-eighth of an inch above the enamel margin there was a small rounded opening. The fleshy tissue projecting from the opening was extremely sensitive and proved to be connected with the pulp. Under cocaine anæsthesia a canal leading to the pulp cavity was detected.

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[1] *Die Vierteilung der Gefässe in den Zähnen des Menschen* (Anatomische Hefte), H. liv.

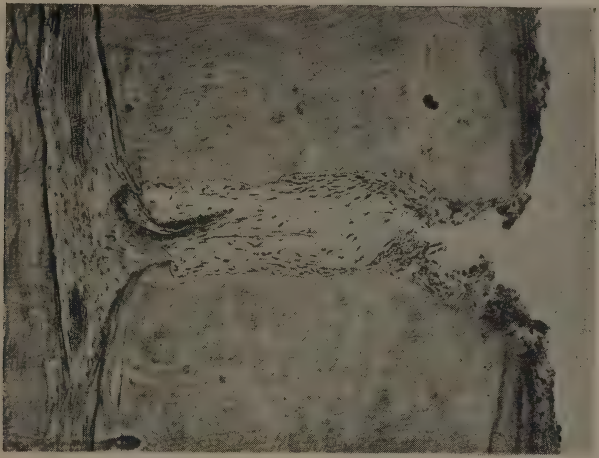
FIG. 470.—Higher magnification of fig. 469. (E. S.)

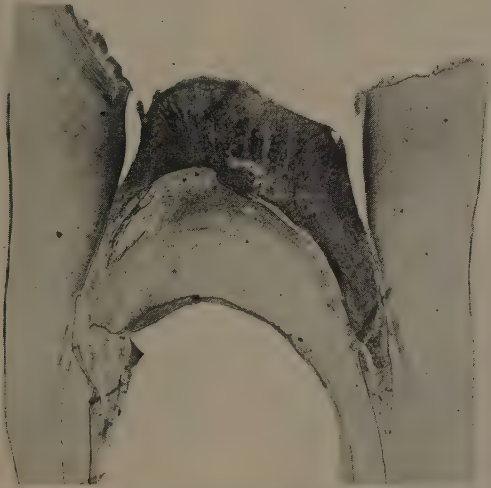
FIG. 471.—Aberrant opening from pulp to periodontal membrane, not of developmental origin. (E. S.)

(C) REGRESSIVE CHANGES.

(1) Fibrous Degeneration.—Fibrous degeneration of the tooth pulp has hitherto been thought to be common, but it is now known that the appearances described were produced by error in histological technique, the error being the lack of access of fixing media to the pulp before post-mortem changes had taken place, a difficulty which may be got over by removing the apex of a tooth and so giving the medium access to the pulp when the specimen is put into it.

Nevertheless fibrous degeneration does occasionally, but rarely, occur. Fish[1] has illustrated such a case which occurred in the maxillary first incisor of a male aged 27, who 12 years before had received a blow on that tooth. The tooth as a whole (fig. 472) shows extensive areas of rarefaction and absorption from the pulpal aspect, and in different areas of the pulp fibrous degeneration (fig. 473)

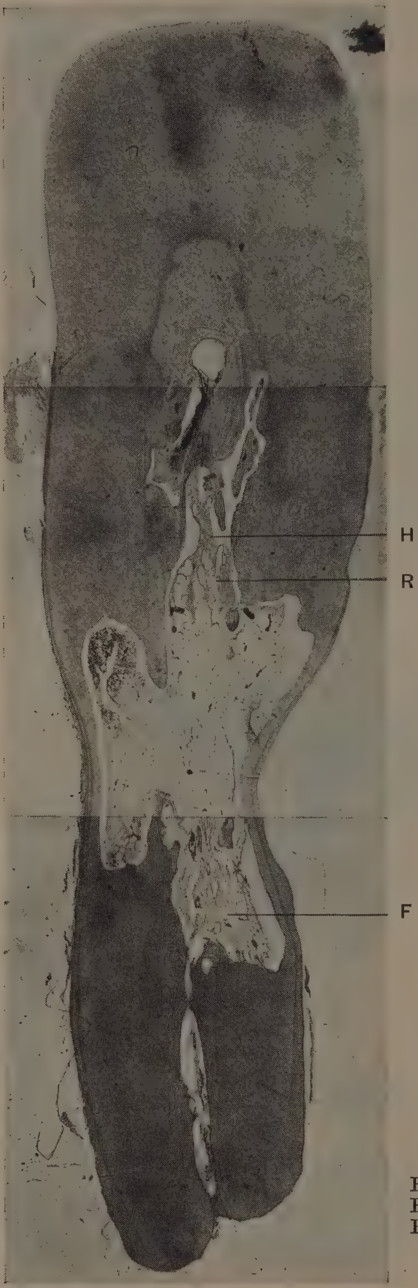

Fig. 472. × 8.

F. Fibrous degeneration (see fig. 473).
H. Hyaline degeneration (see fig. 474).
R. Hyaline material replaced by fat (see fig. 475). (E. W. Fish.) (From *Proc. Roy. Soc. Med.*)

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[1] *Brit. Dent. Journ.*, 1938, lxv, 631.

and hyaline degeneration (fig. 474), and in parts the hyaline material has been replaced by fat (fig. 475). It will be noted (fig. 473) that in fibrous degeneration the tissue is still actively vascular and that but little of the original structure of the pulp tissue remains, only an occasional spindle cell being seen among the collagenous fibres, and that the odontoblasts have entirely disappeared.

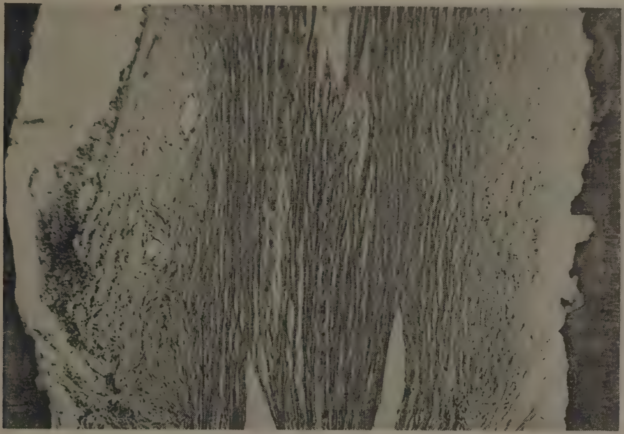

FIG. 473.—Fibrous degeneration. × 94. (E. W. Fish.) (From *Brit. Dent. Journ.*)

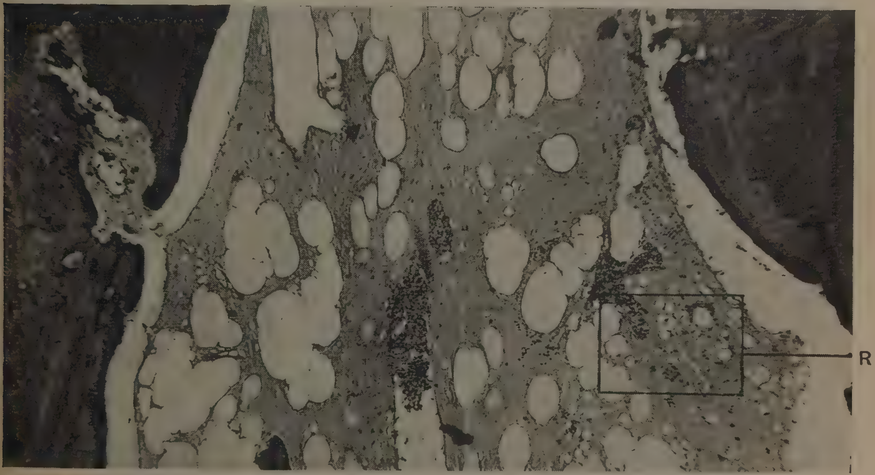

FIG. 474.—Hyaline degeneration. × 100. For R, see fig. 475. (E. W. Fish.) (From *Brit. Dent. Journ.*)

Fish[1] has also produced a true fibrous degeneration in a

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[1] E. W. Fish, *Brit. Dent. Journ.*, 1939, lxvi, 416.

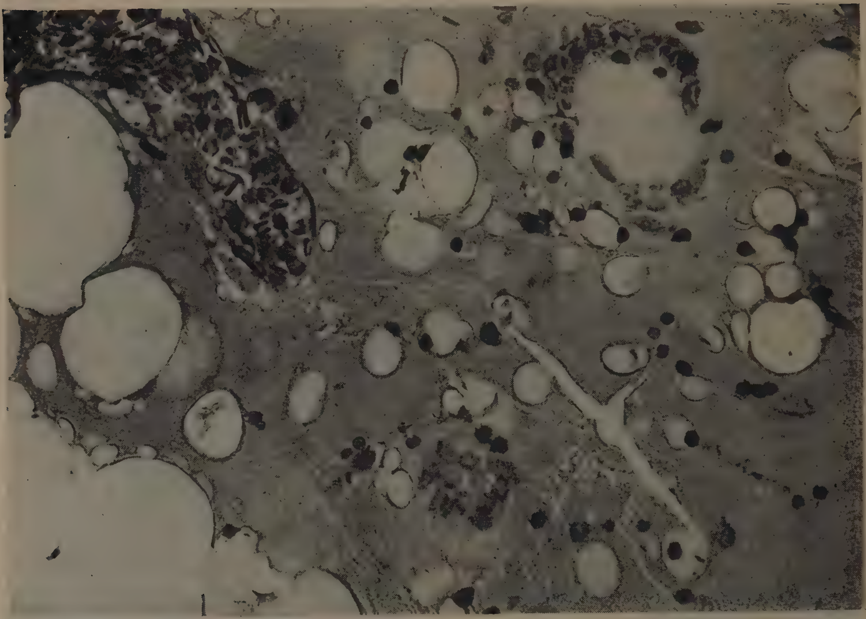

FIG. 475.—Hyaline replacement by fat (in the round spaces). × 611. (E. W. Fish.) (From *Brit. Dent. Journ.*)

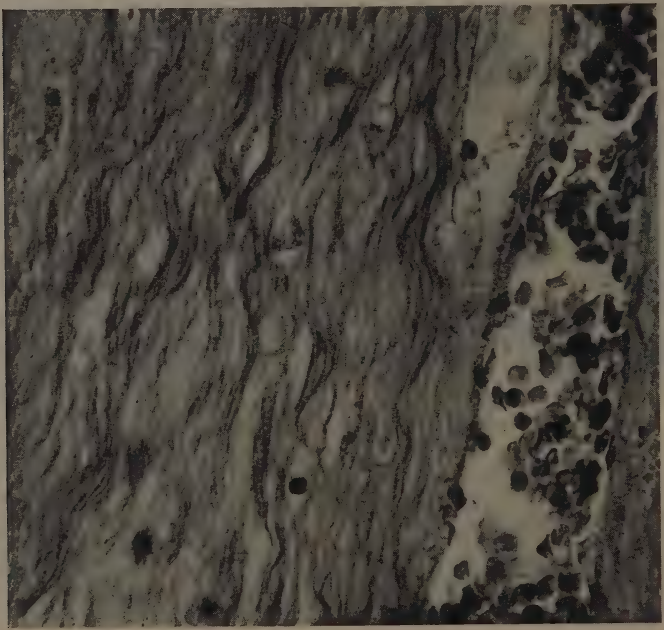

FIG. 476. × 585. (E. W. Fish.) (From *Brit. Dent. Journ.*)

monkey's tooth pulp (fig. 476) after fourteen days' transplantation in the same animal's muscle ; in this case no pulp cells are left, and the pulp tissue is entirely replaced by collagen fibres, which must have been deposited as the fibroblasts degenerated ; he regards this as evidence that collagen deposition is brought about by enzyme action.

(2) **Hyaline Degeneration.**—This is very rare, but occasionally occurs ; it is probably produced by the further degeneration of the collagenous material occurring in fibrous degeneration of the pulp (figs. 472 and 474) and occurs without impairment of the blood supply.

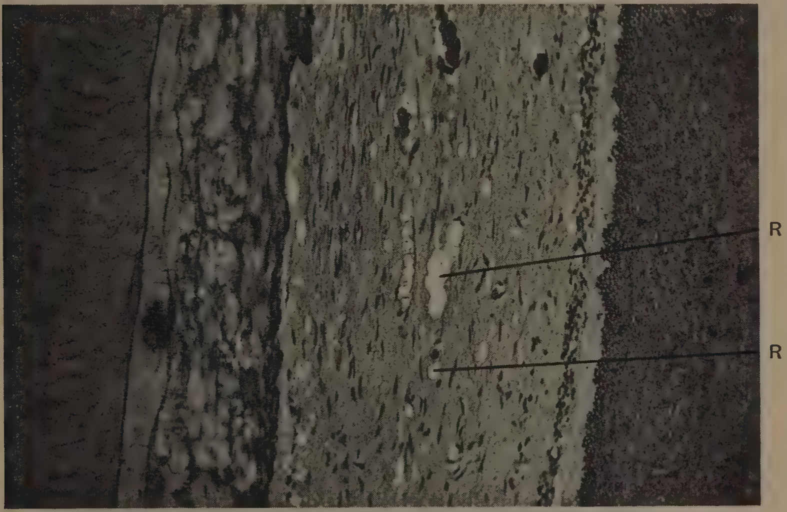

FIG. 476A.—Hyaline replacement by fat at R. × 185. (E. W. Fish.) (From *Proc. Roy. Soc. Med.*)

(3) **Fatty Changes.**—Fatty changes occasionally take place in the tooth pulp (figs. 472, 474 and 475). According to Fish true fatty degeneration does not occur in the tooth pulp, such deposits as occur being brought about by the replacement, sometimes of hyaline material, by fat ; such replacement is also seen in a human molar root in fig. 476A. It will be noticed that the vascularity does not seem to be impaired.

(4) **Calcific Deposits.**—Calcific deposits are almost invariably met with in pulps which have been the seat of inflammatory reaction. These deposits are commonly called ' pulp stones ' (figs.

477 and 478) and are of the same nature as laminated secondary dentine if not identical with it; sometimes they are formed in the substance of the tooth pulp and sometimes near the periphery. It is not uncommon to find in the large pulps of molars that several pulp nodules have become joined together forming a

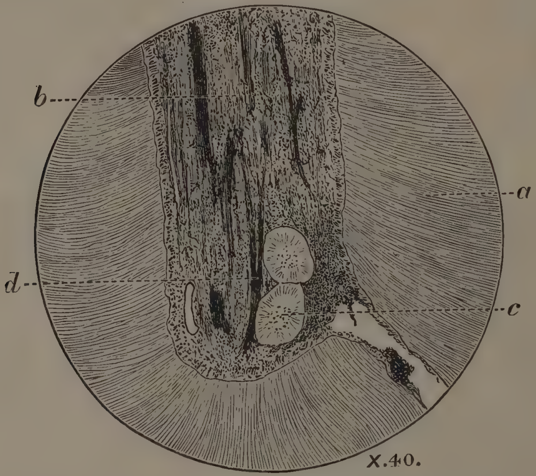

FIG. 477.—Pulp nodules (longitudinal section). (*a*) dentine; (*b*) normal pulp tissue; (*c*) pulp nodules; (*d*) nerve bundles.

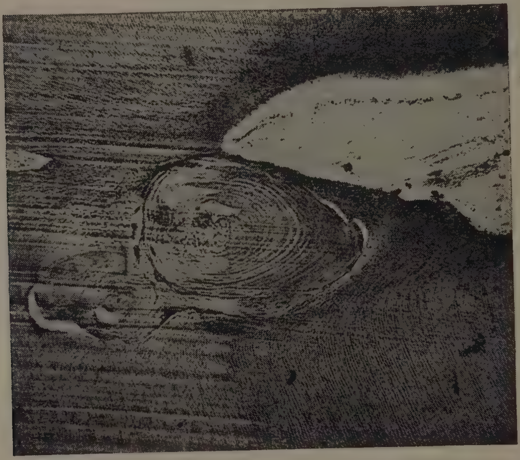

FIG. 478.—Two pulp nodules incorporated in the dentine, the tubules of which change direction to enclose them. (E. S.)

lobulated mass (fig. 479). Calcific deposits are occasionally met with in the healthy pulps of normal young teeth, and in such case can be of no pathological significance.

A. D. Hitchin [1] records a case of a girl, aged 13, who radiologically showed the presence of pulp stones in all the erupted teeth. They gave rise to no signs or symptoms and were only discovered by chance; she showed no other abnormalities. A similar case is recorded by L. R. Weiss [2] in a female aged 24. In both cases it appeared that after the pulp stones had formed further formation of dentine had occurred so as to narrow the pulp canals except at those points where the pulp stones were situated, thereby creating the impression at first sight that the pulp canals had been dilated to hold them. It has been suggested that full or over-dosage with vitamin D may influence their incidence.[3] Westin [4] has recorded that pulp nodules may appear in scurvy in man, and that on healing they may be resorbed.

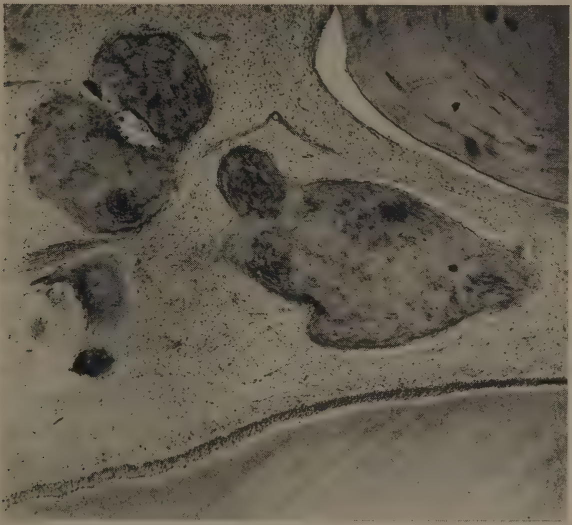

FIG. 479.—'Pulp stones' *in situ*; several are coalesced into the larger mass. (E. S.)

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[1] *Brit. Dent. Journ.*, 1936, lxi, 539. [2] *Dental Cosmos*, 1927, 69, 750.

[3] H. Beck has found that excess of vitamin D produces malocclusion and multiple pulp stones in the teeth of dogs, other dental abnormalities and interferences with growth of the teeth and jaws were also caused. *Journ. Amer. Dent. Assoc.* 1942, xxix, 1947.

[4] Westin, Stockholm, 1931.

The simplest form of calcific deposit is a small pulp nodule. On examination of the tooth after its extraction, these nodules are sometimes visible to the naked eye, and, under a low magnifying power, are seen to be concentrically laminated; in some, not fully calcified, the central portion presents an irregular appearance. Pulp nodules formed near the periphery of the pulp often become included in any secondary dentine that may be formed, the dentinal tubes bending round the nodule. Fig. 477 represents two small pulp nodules from the tooth of a child, aged 14, which was removed for orthodontic purposes. In fig. 478 the pulp nodules have become enclosed by the new formation of dentine. Black considers that deposits of calcoglobulin bear some relation to the formation of pulp nodules.

In pulps which have been irritated or infected there is often a deposit of calcareous material. The deposit may be in the form of small cylinders, which lie parallel to, and are attached to, the fibrous stroma. To the naked eye, the pulp appears stiff, retaining its shape when removed from the canals and resuming its shape after being bent. To the touch, the pulp feels gritty. In advanced stages the cylinders coalesce, being jointed together in an irregular manner. The lumen of the pulp canals is usually diminished in consequence of the formation of secondary dentine.

The whole tissue of the pulp may undergo calcification. To the naked eye, the deposit appears non-nodulated and thus differs from the pulp nodule. As a rule, it is regular in outline and presents a smooth surface. Microscopically, the appearances vary. In some cases the calcified tissue element of pulp is apparent; in others it appears granular on section with a few irregular tubes scattered here and there, or it may exhibit virtually no structure at all.

(5) Avitaminosis A.—Degenerative changes in the nerves of the pulp have been described by King, Lewinsky and Stewart (1938) as occurring in the teeth of rats on a diet deficient in vitamin A, and R. V. Bradlaw [1] has described and figured vacuolation and other abnormal appearances in the human pulp, under this condition, resembling those found in the case of rats.

(6) Avitaminosis C.—For an account of changes taking place in the tooth pulps of guinea pigs in cases of avitaminosis C, see p. 60B *et seq.*

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[1] *Proc. Roy. Soc. Med.*, 1939, xxxii, 1040.

CHAPTER XIV

The Treatment of Pulpless Teeth, open and closed Focal Infection, and the Operation of Apicectomy.

THE treatment of pulpless teeth is at the moment a controversial one and likely to remain so for some years. It is of importance for several reasons, but the most important one is that so frequently such teeth give rise to periapical infection and thereby cause one of the most insidious forms of focal infection, and one which was unknown till radiology gave an insight into what was actually happening.

In periapical infection there are frequently no pus-forming organisms present and so there may be no local signs or symptoms ; moreover, if such teeth have been treated conservatively they may be perfectly functional. When so treated they are a greater risk than when untreated, as there is then no possible exit for whatever toxins may be produced and they are perforce absorbed entirely into the circulation, together possibly with some of the micro-organisms which produce them. This type of lesion gives rise to what is known as 'closed infection,' so named to indicate the impossibility of disposal of the by-products of bacterial growth, save by absorption into the circulation, and in contradistinction to 'open infection,' such as occurs in infected parodontal pockets from which the by-products may escape into the mouth.

Periapical infection shows in a radiogram as a radiolucent area around the apex of a root (see fig. 486). All radiolucent areas are generally taken as indicating that infection is present, and indeed, according to A. Bulleid all pulpless teeth have periapical infection present, usually *Streptococcus viridans*, but occasionally a hæmolytic streptococcal infection may be present. It is known that leakage of arsenic and formalin through the apical foramina may cause rarefaction of periapical bone ; other drugs also so used are often powerful and penetrating and there is every likelihood that they could cause it, but we have no exact knowledge on this subject. One must admit that very many people have had several such periapical lesions present in their mouths, perhaps for many years, without any consciousness on their part that harm has accrued therefrom, but that such is often not the case will be found on perusal of Chapter XXV. These lesions are found most frequently in people who take every care of their teeth, the reason being that they occur more frequently in filled pulpless teeth than in teeth with dead pulps which are left untreated, though they occur in these latter also.

A pulpless tooth with an infected pulp canal frequently gives no abnormal response to percussion (periodontitis), but the contents of the pulp chamber and canals will always be extremely foul. Without entering into the details of the operative technique commonly followed in the treatment of such teeth, one may say that the object aimed at is the sterilisation of the pulp canal and the periapical tissues.

Operations with this end in view are undertaken daily by the dental surgeon, but the degree of success which attends them (by success meaning sterility of root canals and periapical tissues) is probably very small; yet he should be satisfied that he has attained these ends before he proceeds with the conservative restoration of the tooth.

The only signs that the practising dental surgeon has to guide him that he has attained these desired ends are :—

(i) The cessation of the clinical signs of periodontitis (which may never have been present at all).

(ii) The sense of sight, that the dressings he removes from time to time appear clean.

(iii) The sense of smell, that the foulness has disappeared.

It can readily be grasped how futile these tests are. Our sense of smell is a poor sense, and with the powerful odours of the drugs used for dressing teeth about, is of no value whatever, even to detect gross infection by gas-producing organisms; added to which is the fact, not sufficiently realised, that many infective organisms do not give rise to any smell.

It should be realised that neither the surgeon nor the dermatologist has yet found a means of sterilising the skin, and the difficulty that the dental surgeon is confronted with in sterilising tissue half an inch or more below the surface will then be more fully appreciated.

It must be remembered that living tissues cannot be sterilised by means of antiseptic drugs. The best such drugs may do is to lower the vitality of the organisms (and possibly, as a few of them do, produce a local leucocytosis) so that the leucocytes of the host may be able to overcome them and remove them by ingestion and digestion, but the same drug which injures the infecting organisms will also injure the living tissues in which they exist; the living tissue, however, being nourished by the blood and lymph circulation may recover more rapidly and so possibly bring about sterilisation.

Such an ideal is not often reached in the case of infected periapical areas.

[1] In a series of experiments carried out at the London Hospital some years ago, culture experiments and microscope slide films were made from such teeth after they were thought to be clinically sterile. In most cases, even after weekly dressings extending over six or more months, the dressings were not sterile on removal; two such teeth gave positive sterile tests, each after about six weeks' treatment, and another gave a similar result when weekly dressings had been continued for a year.

The results should be tested by periodic radiograms to see if, as sterilisation is brought about, new bone is deposited in the periapical area, but it is very important that such radiograms should be standardised as otherwise false appearances are easily produced.

The time and expense of such treatment necessarily make it almost impracticable for use by the practising dental surgeon, especially as its results are so inconclusive and disappointing, but one must conclude that when tested by cultures and films such teeth may, usually after prolonged treatment, be rendered sterile.

There can be no doubt that the safest and most rapid means of getting rid of such periapical infection is by the removal of the teeth; many patients will object to such treatment as being too drastic, particularly when it affects one anterior tooth in an otherwise complete denture, and many dental surgeons will object on account of the comfort and restoration of function and appearance which they can afford to such teeth by conservative means. On the other hand, it is a fact that periapical infection is but rarely removed by conservative measures, and when present is always a potential source of infection elsewhere. The possibilities of this should always be explained to the patient and the onus of the retention of such teeth must be borne by the patient.

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[1] *Lancet*, ccxi, 1926, 2, 300. (E. S.)

It will be obvious that in the case of multirooted teeth, particularly when the root canals are tortuous, narrow, or partly occluded by calcific deposits, the difficulties of sterilisation will be proportionately increased, and, in addition, the work of Hess has shown how frequently there are branching canals from the pulp (fig. 480) and multiple foramina at the apex (fig. 481), of which we have no accurate knowledge when attempting sterilisation. The occurrence too of an extra root, sometimes of diminutive size, is by no means uncommon, and aberrant openings into the pulp canal also occur, but we do not know how commonly (see figs. 48, 469, 470, 471 and 545) and radiography will not necessarily give evidence of either of these.

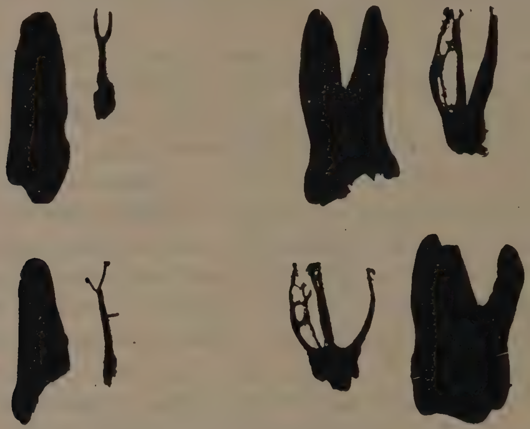


Fig. 480.—Pulps of premolars and molars.
(From Parfitt's 'Operative Dental Surgery.' Edward Arnold and Co.)

Granted freedom from these difficulties and abnormalities, more success appears to have attended recent efforts in the conservative treatment of teeth with periapical infection. Apparently the best method in vogue is that of filling the pulp canal with copper amalgam after it has been rendered as clean as possible by mechanical and chemical means, and any discharge from the apical region has ceased. The copper amalgam appears to encourage and maintain sterility of the periapical area and is apparently a great step forward in the treatment of the condition; radiograms also appear to show that resolution is so good that new bone may be deposited in the previously rarefied and infected areas (see figs. 482 and 483). According to C. S. Henderson, who has studied this technique extensively:

'Preliminary root canal treatment consists of drainage of the apical tissues followed by cleaning and enlarging of the root canal, carried out under aseptic conditions. As the root canal can be regarded merely as a receptacle for seepage from the apical tissues, the procedure is governed by the condition of the canal at the time treatment is commenced. In order to facilitate instrumentation access to the canal should be obtained by opening through the cingulum. In the case of a dry canal, cleaning is commenced immediately with files and barbed broaches, several visits being necessary to clear the canal to the apex. Following this, enlargement of the canal is obtained with hand reamers, large reamers of increasing size being used to avoid perforation of the apex; during this stage the root is radiographed with a diagnostic wire in position to ensure that enlargement is carried to the extremity of the canal. Adequate enlargement of the root canal is considered a very essential part of the technique. It is then washed out with hydrogen peroxide introduced in a hypodermic syringe,

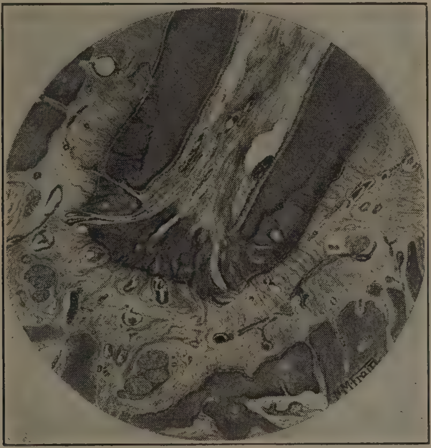
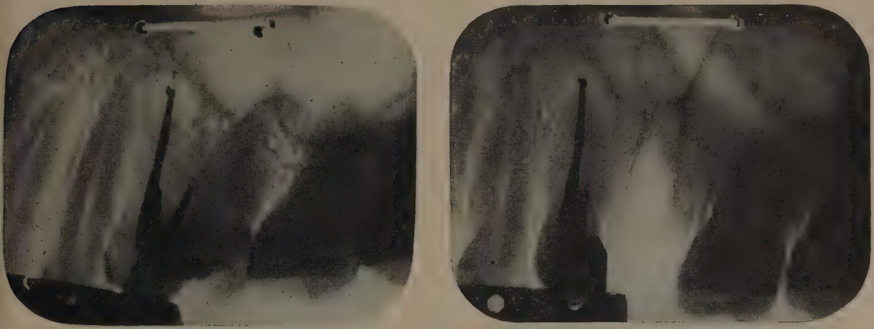


FIG. 481.—Section of tooth showing multiple foramina. (N. G. Thomas.) (*Journ. Nat. Dent. Assoc.*)

care being taken that the point of the needle is not introduced so far into the canal as to become wedged in the apical foramen. Finally the canal is dried out and is then ready for the insertion of the root filling. In cases where a discharge is present on opening up the root canal drainage must be maintained until this has subsided. It is necessary at the beginning to open up such canals at 24-hour intervals to evacuate the pus, gradually increasing the interval between visits as the discharge decreases. Eventually instrumentation of the canal is proceeded with as already described. Providing the canal is managed in this way a flare up is seldom provoked and the crown cavity can be sealed up with a cement between visits and not left open to the mouth, which should be regarded as an undesirable practice. In certain cases prolonged drainage fails to control the apical seepage and the only satisfactory way of dealing with such cases is to clean out and enlarge the canal, insert the root filling and perform a root resection in one visit. The use of caustic antiseptics in root canal treatment seems to be entirely empirical and their passage into the apical tissues has an obviously deleterious effect. No permeability seems to exist between the dentine and cementum in the pulpless tooth, and in consequence, it is doubtful if these caustic drugs even penetrate, quite apart from sterilising the cementum which is the eventual nidus of infection.

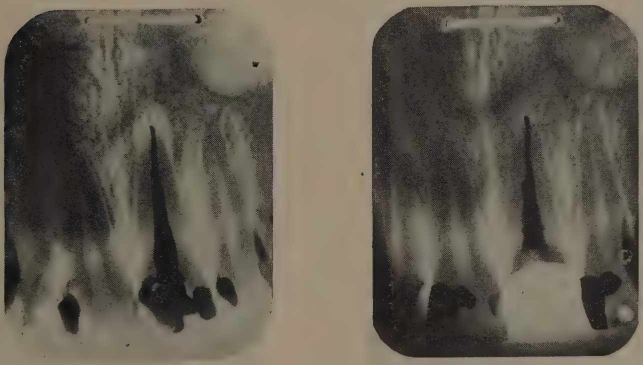
‘ The procedure for inserting the root filling is as follows. The distance from the apex to the incisive edge of the tooth is marked on a root canal plugger. The copper amalgam is prepared and very small pieces are introduced into the canal with long beaked conveying forceps. If large pieces are used there is always the risk that they will become wedged in the canal before reaching the apex. The filling is worked to the apex with the root canal plugger, and for this purpose it is best to use a fine plugger, keeping the plugger point applied to the walls so it is worked round the circumference of the canal. Further small pieces are inserted and the same procedure adopted, until it is shown by the fact that the plugger cannot be introduced so far into the canal that approximately the apical third has been filled. In the case of a root canal with an open apex, the distance from the apex to the incisive edge has first to be obtained by means of a diagnostic wire and the length recorded on a root canal plugger, which should be sufficiently stout in this case to wedge short of the apex, so lessening the chances of pushing the filling through the apex. Actually if the filling does penetrate the apex it does not seem to have any irritating action on the tissues.' (See figs. 482 and 483.)

When pus-forming organisms are present in periapical infection it occasionally happens that at some stage the periapical tissues are stripped from the apex, and the bared portion, by losing its vital connection and being impossible of access, cannot be sterilised, and so maintains infection. Frequently such infection is accompanied by the formation of a sinus opening on to the surface; there is, in effect, a chronic abscess with a portion of necrosed tooth apex maintaining infection. Under these circum-



January 1933. February 1935.

FIG. 482.—Root canal treated and filled with copper amalgam. (C. S. Henderson.)



July 1932. November 1933.

FIG. 483.—Root canal treated and filled with copper amalgam. (C. S. Henderson.)

stances amongst others the operation of apicectomy has been devised for the sterilisation of the apical area.

The Operation of Apicectomy.—Though this operation has been practised for over thirty years, only of late has its general adoption in selected cases been advocated. Until recently radiographic evidence had not been produced demonstrating the post-operative healing of the periapical tissues, and particularly the replacement of bone.

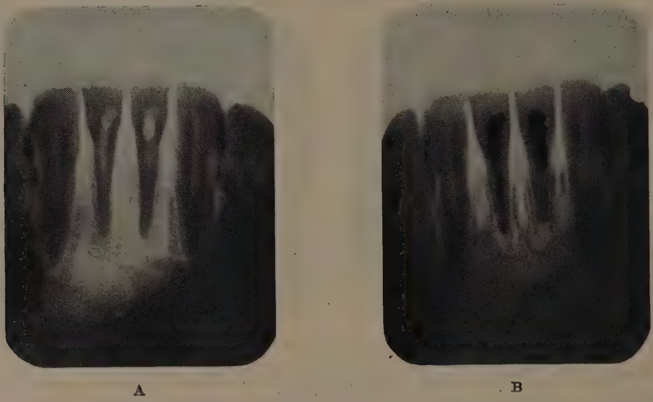


Fig. 484.—Pulpless teeth: A. Before treatment. B. After apicectomy, showing new bone formation. (G. T. Hankey.)

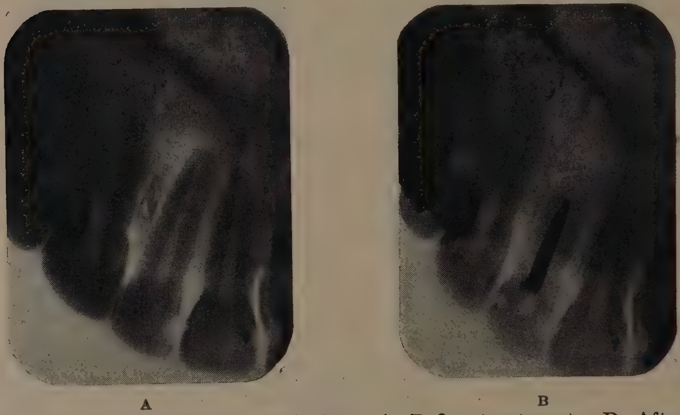


Fig. 485.—Pulpless maxillary incisor: A. Before treatment. B. After apicectomy, showing new bone formation. (G. T. Hankey.)

W. E. Herbert[1] and G. T. Hankey (figs. 484 and 485) have shown, by means of standardised radiograms before and after operation, that complete healing and the formation of new bone may follow a successful apicectomy both in the maxilla and mandible. (The formation of new maxillary bone, under other circumstances, was demonstrated by E. B. Dowsett in 1931. See p. 699.) If the cases are carefully selected, the contra-indications rigidly adhered to, and the technique and skill of the operator of requisite degree, the prognosis is excellent, and justifies the retention of teeth treated in this way. The scope of the operation is limited to the maxillary six and mandibular four anterior teeth when they would otherwise have to be sacrificed as a result of periapical infection ineradicable by the ordinary methods of root treatment.

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[1] *Brit. Dent. Journ.*, 1937, lxii, 137.

Radiographically, the most favourable type of apical osteitis for operation is that which is sharply defined (fig. 486); diffuse areas of rarefaction are not localised and are therefore less favourable. Incidentally, all teeth undergoing ordinary root treatment should be radiographed both before and after, and those having curved apices which cannot be reamed out and efficiently treated and completely filled should be excised by this technique.

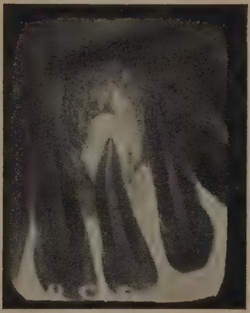
Fig. 486.
(G. T. Hankey.)

The contra-indications for apicectomy are: the infected pockets of parodontal disease, large areas of osteitis the eradication of which would necessitate the removal of more than the apical third of the root, and a predisposition of the patient to systemic streptococcal infections.

Stewart Ross,[1] working on dogs and monkeys, advocates the operation for the following reasons: he states that though in youth there is passage for fluids from the pulp to the periodontal membrane throughout the whole of a root, in adult life this is only true of the apical third, and later on even this is not permeable except through accessory root canals; inferring that permeability to fluids also implies permeability to infection, he states that in an adult tooth the whole of the infected portion of tooth may be removed by taking the apical third; he also states that the epithelium of the gum tends to grow into the wound and attach itself to the exposed cementum. This question of the impermeability of cementum does not seem to be altogether clear, as MacGregor's findings concerning lymphatic flow seem somewhat to contra-indicate its impermeability (see p. 585 *et seq.*), at all events in young teeth.

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[1] *Brit. Dent. Journ.*, 1935, May 15.

The technique employed by G. T. Hankey is along the following lines :—

Before the operation is commenced the rubber dam should be applied, all caries and infected material removed, the root canal reamed out to the apex and dressed at frequent intervals ; debris should be expelled by the interaction of dilute sulphuric acid with sodium peroxide, hydrogen peroxide being liberated by this means ; finally, dressings of pure tricresol or dichloramine T are used, after which the canal is dried and filled with a paste made of zinc oxide and zinc sulphate powder with oil of cloves and carbolised resin, this being forced well home with well condensed gutta-percha. The apicectomy should be performed immediately afterwards, or alternatively the pulp canal can be filled with copper amalgam and the apicectomy performed the following day. Two tablets of veramon are given a quarter of an hour before operation and sterile towels placed over the patient's chest and hair. A submucous injection of 2 per cent. novocain is given, $1\frac{1}{2}$ c.c. buccally and $\frac{1}{4}$ c.c. palatally, using as little adrenaline as will ensure good vision. Ten per cent. cocaine hydrochloride on a gauze strip is placed on the floor of the nose if the area of infected bone is in close relationship.

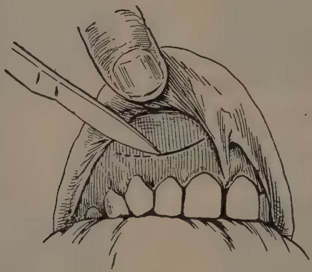

FIG. 487.—Incision for Apicectomy.
(F. St. J. Steadman.) (From the *Dental Record.*)

A curved incision (fig. 487) is made through the gum and periosteum overlying three or four teeth, having its convexity towards the crowns of the teeth and not approaching nearer to the gingival margin than one-third of an inch, so as to lessen the risk of necrosis of too thin a band of gum at the gingival margin. The base of the flap so outlined is thus towards its blood supply. The gum and periosteum are then raised from the bone and held retracted by an assistant. A window is then made in the bone to expose the apex it is desired to remove; this is done with a gouge and mallet, a trephine on the dental engine, or by making four small limiting perforations with a rosehead bur and joining them up with a fissure bur, or, if the bone is soft, an enamel chisel may be used. The lower border of this window must not be nearer to the crown than the line of amputation of the root. Having exposed the apex, a tapered cross-cut fissure bur is used in a saw-like manner to resect the root end; more than one-third should never be removed (fig. 488).

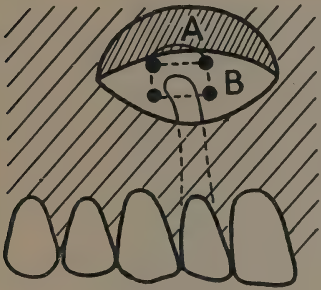

FIG. 488.

A. Under surface of gum flap retracted upwards.

B. Surface of bone exposed, showing method of cutting window with rose head and fissure burrs.

(G. T. Hankey.)

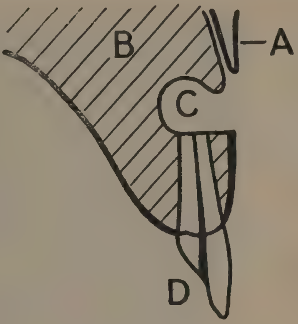

FIG. 489.

A. Gum flap retracted.

B. Bone.

C. Cavity after apex and diseased bone have been removed.

D. Tooth, showing cut root-end flush with floor of cavity.

(G. T. Hankey.)

The most important part of the operation has now to be done; this consists of the thorough removal of all infected periapical bone, and is done by means of large rosehead burs or with a small Volkman spoon. The cut surface of the root end is then treated with silver nitrate, which is reduced with a hot burnisher. The cavity is then gently washed out with a mild antiseptic followed by normal saline. The cut root end must be flush with the floor of the cavity (fig. 489).

The flap is then pressed back firmly into place, secured by one horse-hair suture and tincture of iodine applied to the gum.

Subsequent pain is rare and usually there is only superficial soreness.

So far the problems concerning the treatment of pulpless teeth have only been dealt with as they concern known periapical infections, either with or without pus-forming organisms. There still remain :—

(i) Teeth with infected pulp canals, but apparently no periapical infection.

(ii) Teeth with living, but exposed and infected, pulps which it is necessary to devitalise.

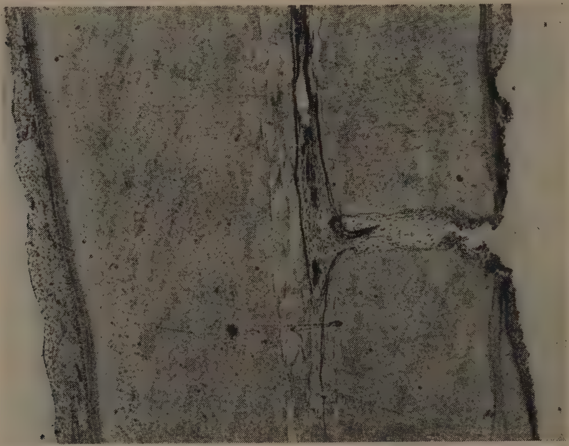

Fig. 490.—Aberrant communication between pulp canal and periodontal membrane; one cornu of the pulp was exposed. Mandibular permanent first molar. (E. S.)

Concerning the former, enough has been said to indicate what care should be taken and what the risks are of producing periapical infection by one's manipulations should one decide to treat such teeth conservatively. Concerning the latter, it is obvious that the risks are less, but even so, they are by no means small.

There is an important factor which has never received the consideration it merits, and which is intimately concerned with the treatment of pulpless teeth : it is the occurrence of *aberrant openings into the pulp canal* (fig. 490). (See also figs. 48, 471 and 545.) Histological experience informs one that these are of more frequent occurrence than is ordinarily supposed ; one can form no idea as to their presence beforehand, and they are often so small as not to be seen on the extracted tooth with the naked eye and occur on any aspect of the attached portions of teeth, which appear normal in all other respects. Though such openings occur on apparently normal teeth they seem to appear more often under pathological circumstances. As one has no knowledge of their presence beforehand it is obvious that the contents of these channels, if infected, can bring to nought the most exacting treatment of pulpless teeth which may be practised; and, if such treatment does away with all signs and symptoms of infection (such as they are), so create a false sense of security in that ‘ closed infection ’ may still be present.

The problem of the treatment of teeth with infected pulp canals and periapical tissues is still an unsettled one, and further standardised research work is necessary before it can be claimed that absolute success has been attained. The risks attendant on the presence of any closed form of focal infection are considerable and insidious. On these accounts it is better under present circumstances not to retain pulpless teeth in the mouth, but patients often insist on it; the dental surgeon should explain the pros and cons to his patient—and do his best, but the onus of responsibility should be on the patient.

In very many cases these infections give rise to no signs or symptoms though they constitute an overload of infection which it is unnecessary for the patient to carry. It often happens that when, owing to decreased resistance on account of age or some other cause, the load becomes too heavy, signs and symptoms first become noticeable, possibly after the infection has been present for many years.

Unless, therefore, one can justify the sealing into the body of organisms of streptococcal and other types of unknown virulence, one can hardly justify the conservative treatment of teeth with periapical infection as such treatment at present stands, except perhaps in the case of single-rooted teeth of easy access.

The fact that the trauma of mastication may cause a bacteriæmia when these infections are present is very important, and should always be taken into account when the conservative treatment of pulpless teeth is undertaken. (See also pp. 620, 621.)

These infections are of more gravity in the young, but even so are always potentially harmful factors—such diseases as endocarditis, fibrositis, etc., may have origin from them. (See Chapters XXV and XXVI.)

CHAPTER XV

Diseases of the Periodontal Membrane.

Acute Local Periodontitis—Chronic Local Periodontitis—General Periodontitis — Regressive Changes — Necrosis of Teeth — Granulomata—Anchylosis of the Teeth to the Jaws.

(A) LOCAL REACTION TO INJURY: INFLAMMATION —PERIODONTITIS.

The local adaptive changes resulting from injury to the periodontal membrane are generally known as 'periodontitis.' Two grades of inflammation may be recognised, namely, acute and chronic. In acute periodontitis, the development of the inflammatory processes is rapid, and is accompanied by marked clinical symptoms; in chronic periodontitis, the reaction is slow in development and the clinical signs are either very feebly marked or absent altogether.

(1) Acute Local Periodontitis.

Causes.—Acute periodontitis may arise from: (*a*) *trauma*, too rapid separation of teeth preparatory to filling; the application of too much force when teeth are receiving orthodontic treatment by mechanical appliances; or the passage of an instrument into the membrane during the treatment of the root canal. (*b*) The commonest cause is injury from the *toxic products of bacteria* which reach the membrane via the pulp canal or the gingival margin, a good example of the latter being seen in cases of suppuration in connection with partially erupted mandibular third molars. (*c*) Injury to the membrane leading to acute periodontitis may also arise from the injudicious use of strong *chemical agents* in the treatment of the pulp canal.

Generally speaking, the reaction set up by injury from chemical agents or trauma terminates in 'resolution,' whilst the reaction started by the toxic products of bacteria may end in suppuration.

Morbid Anatomy and Pathology.—The primary effect of the injury is to cause a temporary contraction of the blood-vessels, and this is succeeded by a slow but progressive dilatation. The increased flow of blood to the part and the resulting inflammatory œdema cause the tooth to be slightly raised from its socket, and the result shows itself clinically by the tooth appearing to be too long. The further development of the inflammatory process consists of a slowing of the blood current accompanied by increased outflow of lymph from the vessels and migration of the leucocytes. If the injury is not of a bacterial character, the reaction may not reach beyond this stage, and the fluid exudate passes into the lymphatics, the debris of any dead tissue is removed by the leucocytes and the parts return to their normal condition.

When the injury is bacterial in character, the inflammatory process is characterised by a marked attraction to the part of leucocytes of the polymorphonuclear variety accompanied by a rapid liquefaction and digestion of the tissue elements. A zone of tissue around the tooth is thus destroyed and its place is taken by pus. Adami [1] points out that in suppurative inflammation there is a period of incubation during which the bacteria multiply and the migration of the leucocytes to the part does not occur until after the cells of the part have become swollen and the vessels full of blood. The delay in the attraction of the leucocytes is, he thinks, due to the fact that some little time must elapse before sufficient toxins are formed by bacteria to attract the leucocytes. Clinically this is shown about the second day by the rapid development of symptoms following the onset of a septic periodontitis. The bacterial growth continues until the organism is able to offer sufficient resistance; pus, consisting of liquefied tissue and dead and living micro-organisms and leucocytes, is then formed and is walled in by a dense layer of leucocytes, and an abscess is formed. When pus has formed it may: (*a*) become circumscribed, and the condition become more or less chronic; (*b*) find its way towards the surface and point; (*c*) burrow along the fascia and the muscle sheaths.

When pus becomes circumscribed, the inflammatory process subsides and a zone of granulation tissue forms around the pus and encloses it. The abscess may then remain inert, but more commonly the suppuration slowly spreads and gives rise to a type of chronic abscess.

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[1] 'The Principles of Pathology,' 393.

When pus approaches the surface, it follows the direction of least resistance. It may escape at the gingival margin, but in most cases it finds its way through the thinner outer alveolar wall and points on the gum over the affected tooth. As long as the pus is confined in the bone, the muco-periosteum is only slightly œdematous, the cellular tissue of the face being unaffected. But when the pus has gained an exit through the alveolar process a rapid swelling of the cellular tissues takes place owing to a diffuse cellulitis. The extent of the swelling is determined by the virulence of the organisms producing the suppuration and by the resistance of the tissues. The abscess may discharge spontaneously, and, if the tooth or source of infection be removed, healing by granulation usually ensues. In the event of the source of infection remaining, the surface is kept constantly infected and a chronic sinus results.

When the pus burrows along the fascia and muscle sheaths, it may lead to troublesome complications.

In the maxilla suppuration around the maxillary first and second incisors may spread into the nasal fossa. Pus from the region of the maxillary second incisors is prone to burrow under the muco-periosteum of the hard palate and form a large fluctuating swelling which points either at the junction of the hard and the soft palate or in the hard palate. Although this condition occurs most frequently in connection with the second incisor, it also arises from the palatal roots or the second premolar or first permanent molar, and indeed may arise from any tooth in the maxilla. The pus may take a direction towards the maxillary sinus, invade this cavity and lead to suppuration in it. This occurs generally in connection with the second premolar or the molars, but may arise from any tooth in the maxilla. Infection from any maxillary tooth may track upwards to the orbit and set up an orbital periostitis and cellulitis.

Infection around the maxillary molars may spread through the posterior dental veins to the pterygoid plexus and from the latter infection may pass by way of the emissary veins to the cavernous sinus. This type of case exhibits the following clinical features: Pain around the tooth with signs of suppuration in the early stage; the infection spreads rapidly and the eyelids become swollen and œdematous; severe pain in the head which becomes extreme with the slightest movement; the patient loses consciousness and passes into a condition of stupor; the head is drawn to the affected side; there may be bilateral ocular proptosis, the temperature rises considerably, and there is increase in the pulse and respiration rate. The prognosis is bad. Pus around the canine may burrow upwards towards the inner canthus of the eye, and in one case seen an abscess in connection with the maxillary first premolar pointed near the angle of the mouth.

In the mandible, pus, instead of finding its way through the outer alveolar plate, may penetrate the inner plate; this usually happens in connection with the third molar, because the inner alveolar plate is thinner in that region, or it may extend into and open on the floor of the mouth. From the incisors and canines, pus, especially if it penetrates the outer alveolar plate low down, may strip up the periosteum and eventually open under the chin. The insertion of the buccinator will usually influence the direction taken by the pus. If pus penetrates the bone outside the attachment of the buccinator, it may burrow into the tissues of the cheek, perforate the skin and open on the face. The most severe form of dento-alveolar abscess arises in connection with the molars. The pus may burrow under the masseter, or under the internal pterygoid and burrow among the planes of connective tissue in the neck, producing a diffuse submaxillary cellulitis (angina Ludovici). The onset of the cellulitis is attended by a marked rise in temperature; rigors often occur, and there is great prostration. There is marked œdema of the tissues forming the floor of the mouth, the mucous membrane being pushed up in such a manner that it frequently suggests a second tongue. The swelling increases rapidly over the front of the neck, spreads backwards to the parotid region, upwards to the orbit and, in the later stages, involves the thorax. There is usually œdema of the larynx. The meninges may become involved by extension of phlebitis along communicating venous branches. The condition, unless treated early, has often been known to terminate fatally. A case of extreme severity which, however, terminated favourably, was recorded by A. Kendrick.[1] The trouble arose in connection with a mandibular left permanent first molar which one year previously had been fractured. The left eye was closed, and pus escaped from the lower orbital margin and from the upper eyelid, a large sloughy opening being present in the centre of the cheek. There were several sinuses along the border of the mandible. The pus had travelled down the anterior border of the sternomastoid to the clavicle, where several sinuses opened; there was also a large sinus over the sternum. Pus had burrowed upwards along the ramus into the temporal fossa, the whole temporal region being œdematous. The tooth was removed; the temporal region was incised and the sinuses freely opened up and packed with iodoform gauze. The patient made a steady recovery.

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[1] *Brit. Dent. Journ.*, October 1900.

Extensive necrosis of the maxilla or mandible may result from alveolar abscess (see Chapter XXX), and in severe cases general infection may occur.

An acute alveolar abscess formed in connection with a deciduous molar may involve the developing successional tooth and cause its necrosis and subsequent exfoliation.

Acute suppurative periodontitis may arise in connection with teeth with live pulps. The two following cases are examples.[1] A small cavity appeared on the lingual surface of the maxillary right second incisor in a girl aged thirteen. The pulp was exposed and a dressing of devitalising material was applied for half an hour, the cavity was then enlarged and a fresh dressing applied. Five days afterwards the patient returned with a large alveolar abscess pointing in the sulcus between the gum and the upper lip; this was opened. The next day the dressing in the cavity was removed and the cavity left open for drainage, the assumption being that the pulp was dead. When seen five days later the abscess was apparently cured, but the pulp was found to be partly alive. The second case was in connection with a distal cavity in a maxillary left second premolar. Remnants of live pulp remained at the apices, and arsenic was applied. A mesial cavity in the first molar was prepared and a layer of softened dentine left over the pulp. A dressing of sandarac varnish was packed into the cavities to press away the gum. After two days the patient returned, and it was found that a large abscess had formed.

Occasionally when there is a ‘pocket’ around a tooth, the exit to the surface for the inflammatory exudate and by-products of bacterial growth becomes occluded by œdema, so that an acute inflammation is superimposed on the chronic condition already present, and a parodontal abscess is produced.

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[1] A. E. Baker, *Brit. Dent. Journ.*, May 1895, 268.

Signs and Symptoms.—In the early stage, the tooth causes the patient discomfort, is slightly raised in its socket, and pressure brings relief. At this period the blood-vessels are in a condition of hyperæmia. The tooth then becomes still further elongated and loose, the gum around it being swollen and painful. The vessels are nearing the condition of stasis, and the surrounding tissues are infiltrated with inflammatory exudate, hence the increased rising of the tooth in its socket. Pressure now increases the pain, because the vessels can no longer be freed of their surplus supply of blood. Each act of mastication under such conditions only increases the pressure on the already hypersensitive nerves. If suppuration supervenes, the swelling of the gum increases, the tooth becomes more loose and the pain dull and throbbing. Finally, a fluctuating swelling appears in the sulcus between the gum and the cheek, the face swells quickly, and, the pus being no longer confined in a dense unyielding structure, the tension upon the nerve-endings is relieved and the pain considerably lessened. Suppuration is usually accompanied by pyrexia.

Diagnosis.—The diagnosis is usually easy. Where suppuration has taken place, the surrounding teeth may have become implicated in the inflammation, but the tooth causing the trouble will be looser than its neighbours and more sensitive to pressure and percussion.

Treatment. *Local.*—The first step is to remove the cause, as far as this is practicable. If the condition is due to the passage of drugs through the apex of the tooth, dressings must be removed, the pulp canal washed out and then dried, using wisps of cotton wool or bibulous paper points for this purpose, as they also act as wicks and will help to remove the periapical serous exudate which may contain some of the causative drug in solution ; dressings of glycerin are useful as, being a hygroscopic substance, it encourages the draining away of more local fluid and so probably more of the causative drug in solution. The application of counter-irritants or fomentations to the gums will also give relief. Where the injury has been caused by instruments in the treatment of the root canal, extraction is the best line of treatment.

When the periodontitis is due to bacterial toxins, the first point to decide is whether the retention of the tooth is advisable. Teeth that are unsavable by conservative operations should be removed. Where there is a possibility of retaining the tooth, the septic matter in the pulp chamber should be removed. Care should be taken in performing this operation, as such teeth are extremely sensitive. The tooth should be kept as steadily fixed as possible. If the tooth is filled, or non-carious, so that access to its pulp chamber would involve long and painful manipulation, access may be made temporarily, so as to relieve tension, by drilling a small hole through the neck of the tooth, but if the root canals have been filled such treatment would be useless. This is an old form of treatment not often used now; the operation used to be known by the name of rhizodontrophy. It can be accomplished with a sharp bur without causing much pain. The pulp chamber should be syringed with an antiseptic solution. Fomentations should be applied to the gum over the root of the tooth, or the gums may be scarified. *If suppuration has occurred*, measures must be taken to remove the pus as soon as possible. The removal of the pus may be hastened by the continued application of fomentations over the situation of the tooth, and, as soon as the pus has pointed, the abscess should be opened. The cavity should be thoroughly irrigated with some unirritating antiseptic solution and drainage provided. The abscess should be dressed at least twice a day, and this is especially needful when it occurs in the mandible, on account of the drainage difficulty. Acute alveolar abscesses generally heal rapidly, especially if carefully dressed. In the case of extensive abscess, the patient must be anæsthetised, the suppurating tracts freely opened, irrigated, and packed with gauze or corrugated rubber for drainage purposes; but this is a subject belonging rather to the domain of general surgery.

If the skin shows signs of being involved in the inflammatory process, it should be dressed with a compress dipped in some mild astringent solution (lotio plumbi). If the abscess threatens to open through the skin, the latter should be supported by covering the surface with a layer of cotton-wool painted over with collodion solution; by this means, aided by free opening of the abscess into the mouth, the opening on the surface can often be avoided. When an opening through the skin is unavoidable an incision should be made at the earliest opportunity, and on no account should the abscess be allowed to burst spontaneously. If this precaution is taken, much disfiguration by scar formation may be avoided. The incision should follow, as far as possible, the fibres of the platysma. A cut across the fibres leaves a gaping scar, whereas when the cut is parallel with them the resulting scar is almost imperceptible.

In opening an abscess connected with a mandibular molar, care must be taken to avoid injuring the facial artery as it crosses the mandible immediately in front of the anterior border of the masseter. In cutting, the knife should be directed towards the bone and not away from it. In opening abscesses in the palate, the anterior or posterior palatine arteries may be injured. The incisions into palatal abscesses should therefore be made, as far as possible, parallel with the course of these vessels.

As soon as the active stage of inflammation has subsided the pulp canals should be treated in the usual way. *Where suppuration is extensive* and the pus shows signs of burrowing in an unusual direction, the tooth should be removed immediately.

The diffuse and acute form of submaxillary cellulitis known since 1836 as Ludwig's angina, though it may arise from suppuration originating in any mandibular molar, usually arises from infection around a mandibular third molar, which may be misplaced or not, but in any case has not a very clear field for eruption, possibly from lack of room (growth) in the mandible. The disease usually starts as a parodontal infection around the crown of this tooth, ingress for the infecting organism being via the point where the anterior cusps may already be showing through the gum ; the inflammatory œdema of the overlying gum closes up the point of ingress so that it is not a means of efficient drainage and a parodontal abscess results, with inability to open the jaws (see pp. 5 and 6). Extraction of the third molar at this stage and efficient drainage will cure the condition, but cases are sometimes not seen till a more serious state has developed, and this is brought about by the tracking and burrowing of the pus as described on pp. 451 and 452, producing at first an acute submaxillary cellulitis and inflammatory œdema of the floor of the mouth, spreading later and infiltrating the tissues around the front of the neck. Treatment at this stage, after removal of the third molar, consists in providing free drainage in the infiltrated neck by multiple incisions, which are kept open by gauze or corrugated rubber drains, and hot fomentations. The infecting organism is said to be a streptococcus in 70 per cent. cases.

An invaluable aid in the treatment of such cases has recently come to hand in drugs of the sulphanilamide class [1] which are of especial value in all acute streptococcal infections. These drugs may be given orally in tablet form or parenterally in solution, and as there are now a number of these preparations on the market the dosage should be according to the instructions issued with the particular preparation used. A child's dose is half, and an infant's one-third, of that for an adult. The drug should be started early, and if the hydrochloric acid content of the gastric juice is low, dilute hydrochloric acid should be prescribed. Apparently their action is prophylactic as well as curative. Their mode of action is not clear, as in the laboratory they appear to have no bactericidal action, nor do they promote phagocytosis. The temperature usually falls rapidly after administration is commenced and the toxæmic symptoms disappear.

The taking of sulphur-containing compounds—such as magnesium or sodium sulphate—is contra-indicated during administration, and if an aperient is needed cascara or liquid paraffin should be given.

On account of the factors which usually give rise to this condition Ludwig's angina is most often seen between the ages of twenty and thirty years.

General.—In the early stages a smart saline purge will assist in clearing up a local periodontitis. If suppuration has taken place, the necessity of attending to the general health of the patient cannot be too strongly emphasised. The bowels should be carefully regulated, and, if needful, a tonic treatment prescribed which, in addition to drugs, should include plenty of fresh air and a nourishing diet.

(2) Chronic Local Periodontitis.

(i) Commencing near the Apex of the Tooth.

Causes.—Chronic periodontitis follows on injuries similar to those leading to the acute form, the difference in the reaction of the tissues being due to the lesser intensity and the persistence of the injury. Many cases arise from infection via the pulp canal. In a few instances, the injury may be caused by the passage of a root filling through the apical foramen or from malocclusion, but excessive use is probably the commonest cause.

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[1] Discovered in 1935 by Gerhardt Domagk. They are reduced in the body to *p* aminobenzylsulphonamide, or more briefly sulphanilamide. Recent preparations are sulphapyridine (M. and B. 693) and sulphathiazole (M. and B. 760).

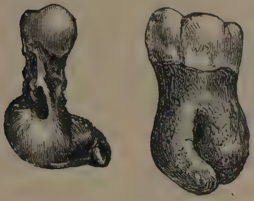

FIG. 491.

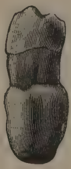

FIG. 492.

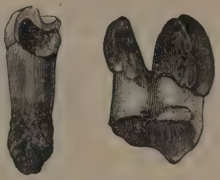

FIG. 493.

It occurs also on retained unerupted teeth, particularly maxillary third molars which, owing to lack of bony growth to allow of their normal eruption, become engaged with the roots of the second molar; the irritation so produced causes an excess production of cementum which joins the two teeth; rarely more than two teeth may become united in this manner (pathological gemination). (See figs. 496, 821, 822 and 823.)

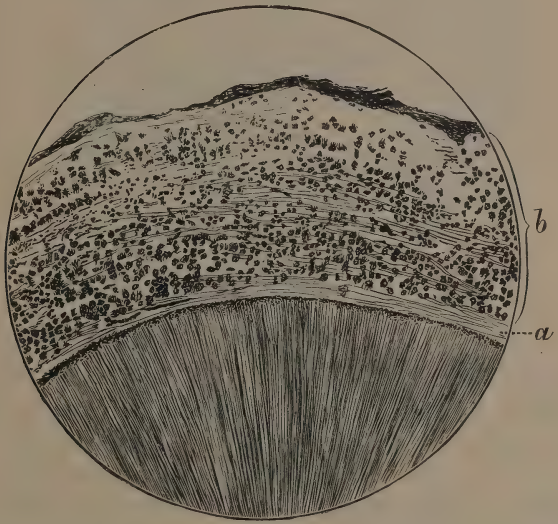

FIG. 494.—(*a*) Original layer of cementum; (*b*) tissue of new formation.

Its occurrence is almost confined to the permanent dentition; rarely it occurs on a retained deciduous tooth.

Morbid Anatomy and Pathology.—Where the injury is of slight intensity the change which occurs consists in a proliferation of the connective tissue leading to a thickening of the membrane, the portion immediately adjacent to the tooth tissue being replaced by cemental tissue. This type of reaction is termed *proliferative periodontitis.* The process is analogous to that which occurs in a proliferative periostitis. The naked-eye appearances of teeth so affected are shown in fig. 491. When the inflammation is continuous and productive, the surface of the new tissue usually presents a smooth appearance, as seen in fig. 492. On the other hand, when the condition alternates between a productive and a rarefying periodontitis, an irregular outline is usually seen, as in fig. 493. Sections through the hard tissues of teeth exhibiting this condition are shown in figs. 494 and 495. It will be noted that in fig. 494 the original layer of cemental tissue is intact, and that the lacunæ of the new tissue are fairly regular, the incremental lines of Salter being well marked. These lines are situated at the junction of the laminæ. The lacunæ in the new tissue are more numerous, and are also slightly larger and coarser than in normal cementum.

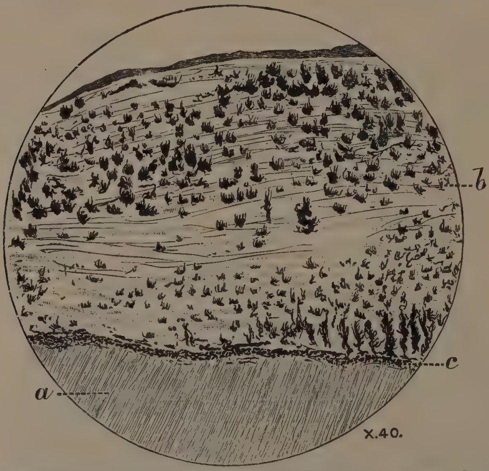

FIG. 495.—Longitudinal section showing new tissue. (*a*) dentine; (*b*) new tissue; (*c*) granular layer.

Simultaneously with the formation of the cemental tissue, a rarefying osteitis occurs, which makes room in the tooth socket for the new tissue. In extreme cases, two or more teeth may be the subjects of proliferative periodontitis; the intervening septa of bone may be completely removed by rarefying osteitis and the teeth become united by cemental tissue. This occurred in the case shown in fig. 496. The condition is known as pathological gemination. (See also Chapter XXII.)

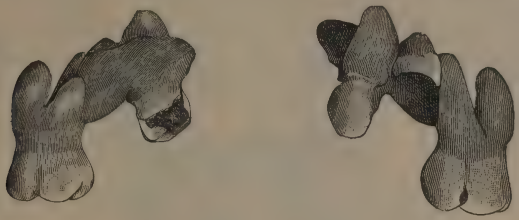
FIG. 496.

The new tissue is often more irregular in character than that shown in fig. 494, the laminæ being absent and the lacunæ placed irregularly (see fig. 497). Vascular canals are at times seen.

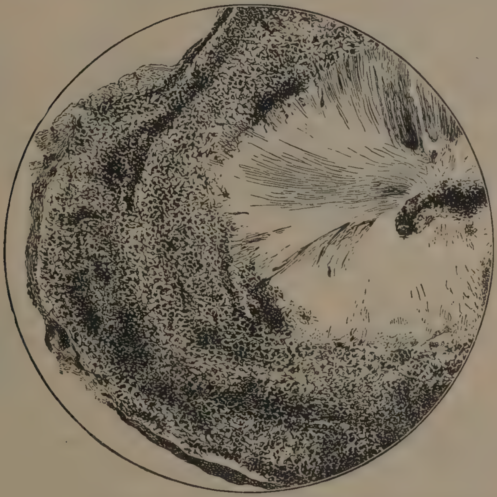
FIG. 497. (Douglas Caush.)

Adami has pointed out that substances which in larger quantities are toxic and lead to degeneration of the tissues often in smaller quantities act as direct stimuli to the cells and to increased growth; that is to say, the increased growth is not secondary to tissue destruction. Similarly, mechanical or toxic stimuli, if not too strong, may result in overgrowth (hyperplasia).

Where the intensity of the injury is greater, the reaction in the tissues is characterised by cell infiltration in addition to the cell proliferation of the connective tissues. Among the latter are multi-nucleated cells (osteoclasts) which absorb the tooth tissue. This type of reaction is termed *rarefying periodontitis*, and the process is analogous to rarefying osteitis. A section through the tooth shows a series of semilunar excavations (Howship's lacunæ) (fig. 498).

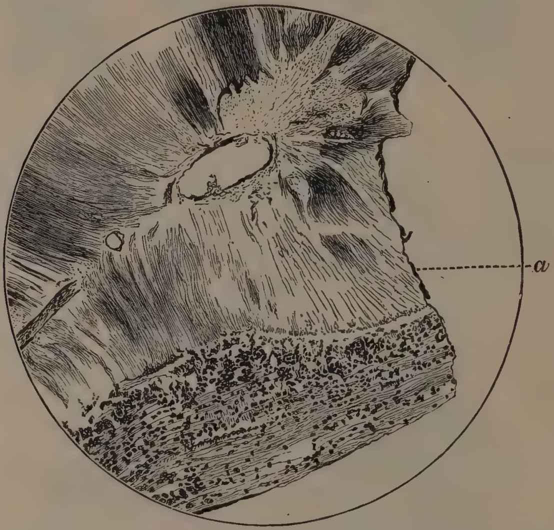

FIG. 498.—(*a*) Margin of tooth showing Howship's lacunæ. (Douglas Caush.)

When active absorption is going on each lacuna may be occupied by a cementoclast (it is rare to find them all so occupied), and in the neighbourhood are to be seen epitheloid cells and small cells, the appearances being in all respects similar to those seen in absorption of the deciduous teeth under normal conditions. As the absorption progresses, the cementum is first removed and then the dentine, and, in rare cases, the entire root may disappear. The difference in the appearance of teeth so affected depends upon the character of the injury; if the injury is intermittent, the absorption is intermittent, and the absorbed surface may be covered with a deposit of cementum in quiescent intervals, as in figs. 499 and 500, or it may be continuous and progressive, as in figs. 501 and 502. The character of the reaction doubtless varies as that of the injury. Teeth showing various stages of absorption are shown in figs. 503 to 507.

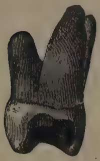
FIG. 499.

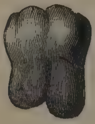
FIG. 500.

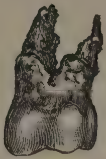
FIG. 501.

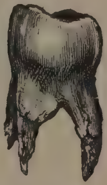
FIG. 502.

Under certain conditions, *e.g.* variations in the intensity of the injury, *the rarefying periodontitis may pass into the stage of proliferative periodontitis* and much of the lost tissue may be replaced, to be, perhaps, again removed if the process resumes greater activity. In other cases, the inflammation may cease altogether and the small-celled infiltration be replaced by cementum. In the case shown in fig. 508 the dentine has been absorbed and replaced by cementum. This specimen therefore indicates the existence of a rarefying periodontitis, followed by the replacement of the small-celled infiltration by cementum, a condition analogous to healing in soft tissue by granulation.

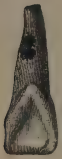
FIG. 503.

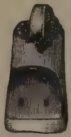
FIG. 504.

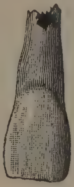
FIG. 505.

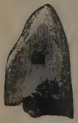
FIG. 506.

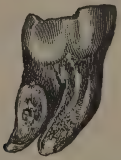
FIG. 507.—Mandibular second molar showing absorption of root due to an unerupted third molar.

In a tooth affected by rarefying periodontitis the apical portion of the pulp canal may become involved and the walls of the canal

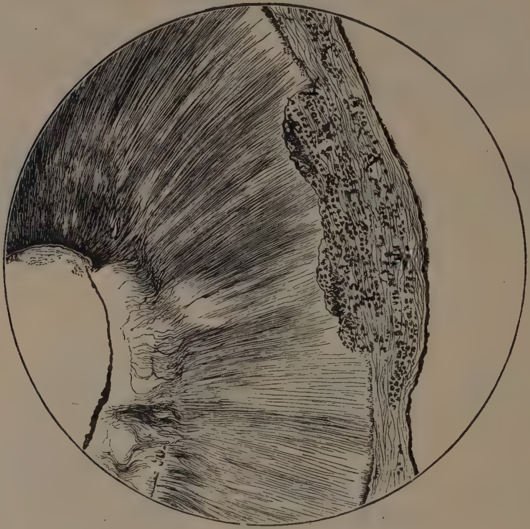

FIG. 508. (Douglas Caush.)

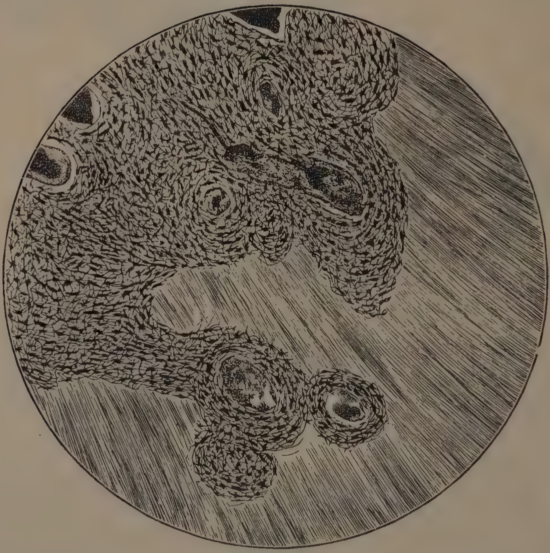

FIG. 509.—Section of a tooth showing dentine replaced by bone.

be destroyed. If the intensity of the toxic process lessens, a proliferative stage may follow and result in the deposition of cementum.[1] In the section shown in fig. 509 the dentine has been replaced by bone. The tooth, a maxillary premolar, was removed from a man aged thirty-three years. It erupted high up towards the outer side of the alveolar process, the root being about two-thirds formed. In another case which came under notice a misplaced unerupted mandibular third molar had been the seat of suppuration. In this specimen there was extensive absorption of the dentine and redeposition of bone.

Hopewell-Smith[2] figures a maxillary first incisor, showing internal absorption of dentine and deposition of bone; and C. S. Tomes[3] records a similar condition in a maxillary premolar which had become loose. The latter tooth is shown in figs. 510 and 511, and sections showing the extent of the absorption of the dentine and replacement by bone in figs. 512 and 513.

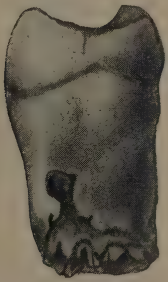

FIG. 510.—View of the exterior of the tooth showing the pitted character of the end of the root with an excavation running a little way up on one side.

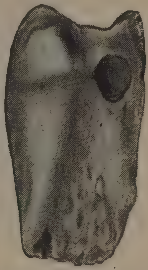

FIG. 511.—View showing position of the carious cavity.

(From *Trans. Odonto. Soc.*)

Chronic injury to the periodontal membrane at times results in changes which are more marked in the surrounding osseous tissue than in the tooth itself. The bone in the neighbourhood of the tooth is removed and its place is taken by a mass of granulation tissue; externally to which there is a layer of fibrosed tissue. These masses of tissue often come away with the tooth when it is extracted (see also p. 489).

When the injury persists and is of a septic or infective type suppuration ensues and produces a condition commonly known as *chronic suppurative periodontitis.* The injury usually reaches the membrane through the apical foramen. The pus formed from the degeneration of the tissues may become more or less confined through being surrounded by a layer of granulation tissue, external to which is a layer of fibrosed tissue, and the abscess thus formed may remain stationary and give rise to no symptoms. More often, however, there is a tendency for the suppurative process to extend slowly, generally in the direction of least resistance, with the result that the alveolar process is destroyed, an opening appears on the gum, and a sinus is formed.

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[1] J. Lewin Payne, *Brit. Dent. Journ.*, 1921, March 15, 235.

[2] 'Histology and Pathology of the Teeth.'

[3] *Trans. Odonto. Soc.*, xxxi, 172.

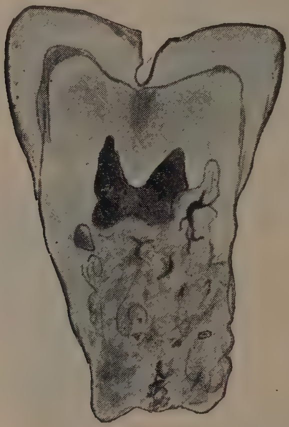

FIG. 512.—Longitudinal section showing the general relation of the new tissue to the pulp cavity.

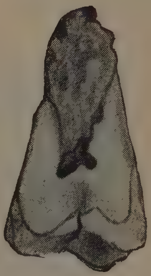

FIG. 513.—View showing the extension of the new tissue upwards into the dentine by the side of the pulp cavity.

(From *Trans. Odonto. Soc.*)

In discussing acute suppuration it was explained at what positions the abscess might be expected to point (see p. 450). The same positions may also be regarded as the probable sites of the pointing of chronic abscesses.

The extent of destruction of the bone which may occur in chronic suppurative periodontitis is shown in the specimens described below.

The mandible of a child about six years of age is shown in fig. 514. The bone around the apex of the anterior root of each molar has been destroyed to such an extent as to form definite cavities ; the outer aspects of these roots throughout their entire length have been denuded of bone. The destruction of bone is more marked in connection with the second than the first molar. The radiogram of the specimen (fig. 515) should be contrasted with that in fig. 516, which is taken from a normal specimen.

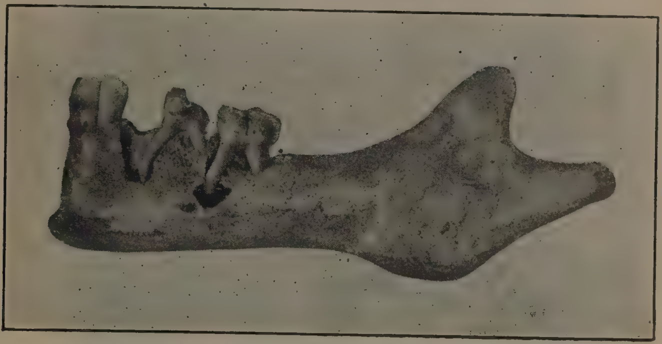

FIG. 514.—Mandible showing destruction of bone around the roots of the deciduous molars. (We are indebted to the publishers of the *Dental Record* for the blocks of figs. 514 to 533.)

If the fleshy covering of this specimen (fig. 514) could be reinstated we should see a marked marginal gingivitis with considerable food stasis, sinuses opening into the sulcus between the gum and the cheek and constantly discharging pus. The child would be typical of many seen at the present time.

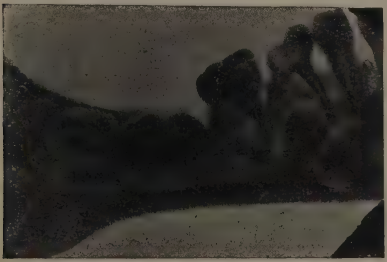

FIG. 515.

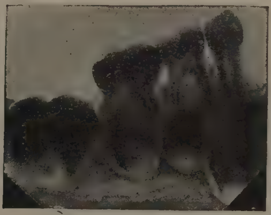

FIG. 516.

A less severe condition is seen in fig. 517. The mandibular right deciduous second molar has been attacked by caries, the destruction of the dentine having at the time of death just reached the pulp cavity. The bone intervening between this tooth and the deciduous first molar has been destroyed and the upper two-thirds of the anterior root has been denuded of its bony cover. There is no appearance of trouble around the apex of the root. The radiogram (fig. 518) of this specimen shows the extent of the bone destruction. It will be noticed that the crypt of the first premolar has been opened on the distal aspect and that the bony layer which should exist between the second premolar and the second molar has disappeared. Put on the covering of soft tissues and we should probably see food packed between the deciduous molars, with the anterior root of the second molar partially denuded, and marked sepsis which would be in free communication with the growing premolars.

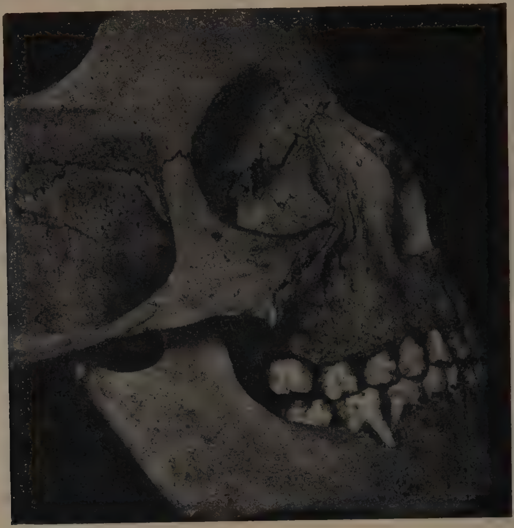

Fig. 517.—Skull and mandible of a child about six years of age, showing the effects on the bone of suppuration in connection with the mandibular right second molar.

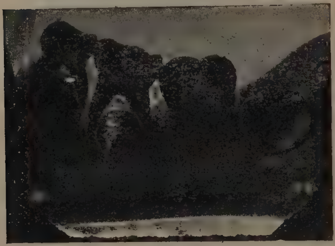

Fig. 518.—Radiogram of the specimen shown in fig. 517.

The maxilla of a child, just over nine years of age, shown in fig. 519, demonstrates that considerable loss of bone may occur. The complete bony covering of the crown of the second premolar has been destroyed, and the posterior aspect of the socket of the first premolar has partly disappeared.

The maxillary permanent incisors, owing to their position, are frequently injured by trauma and it is by no means uncommon to find a transverse fracture just below the neck. If left untreated the pulp is destroyed by sepsis, and infection of the periodontal membrane follows. The clinical appearances are as follows: The remaining portions of the teeth are dark in colour; the dentine is hollowed out and is deeply stained, and there is a

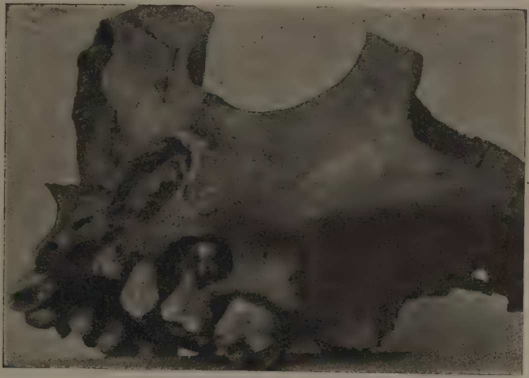

Fig. 519.—Maxillæ from a child just over nine years of age. The crypt of the left second premolar has been destroyed by suppuration.

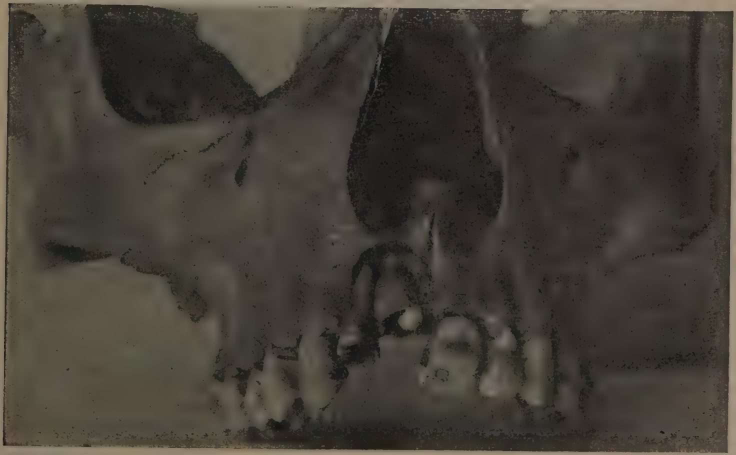

Fig. 520.—Maxillæ showing the effects of suppuration around the roots of the right incisors.

distinct thickening of the muco-periosteum over the roots of the teeth. A sinus may be present and pressure over the region of the root gives rise to tenderness.

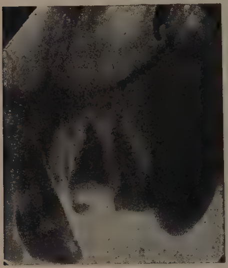

FIG. 521.—Radiogram of the specimen shown in fig. 520.

The morbid anatomy of a case of this type is shown in fig. 520. It is the maxilla of an adult. The right incisors have been fractured—the second incisor through the crown near the neck of the tooth and the first incisor through the root just beyond the neck. The first incisor is little more than a hollow cone, the second incisor, on the other hand, has undergone little destruction. A well-marked cavity is present in the bone, the first incisor only retaining a connection with the bone at the posterior aspect of the root, while the second incisor has the distal and posterior aspects of its socket still intact.

The radiogram of this specimen is shown in fig. 521.

The points to be noted are :—

(1) That there is no well-defined line between the cavity and the surrounding bone, such as is noticeable in radiograms of cysts.

(2) That there is indication of bone posterior to the roots.

A palatal abscess arising in connection with a maxillary second incisor is shown in fig. 523, but before considering in detail the features of the specimen a brief reference will be made to the anatomical relations of the parts involved.

In fig. 522 is a diagram of a vertical section through a maxilla in the region of the second incisor. Attention is drawn particularly to the area of cancellous bone between the posterior aspect of the root and the palatal aspect of the bone. In the recent state, the palatal surface is covered by a dense layer of muco-periosteum. An abscess that appears on the palate must therefore excavate its way through this area of bone, and when it does emerge through

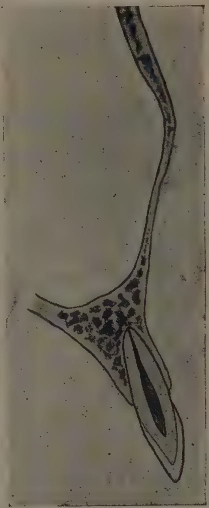

FIG. 522.—Vertical section through the maxilla in the region of the second incisor.

the bone will meet with the muco-periosteum, which is sufficiently resistant to stop actual penetration, with the result that the pus, following the line of least resistance, burrows under the muco-periosteum in a direction towards the back of the palate.

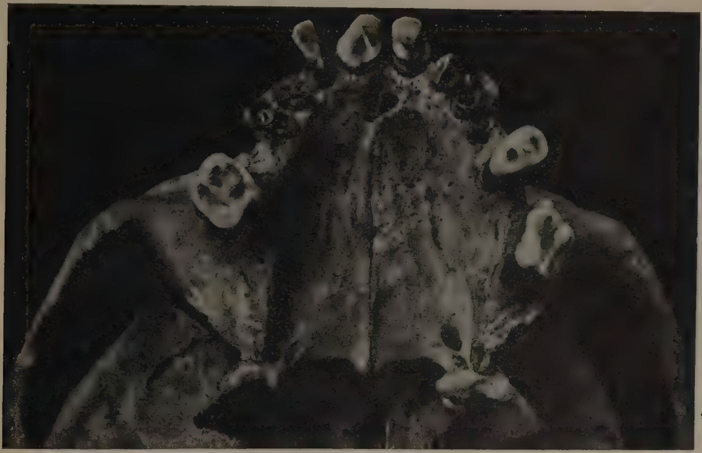

FIG. 523.—Palatal view of an abscess arising in connection with a second incisor.

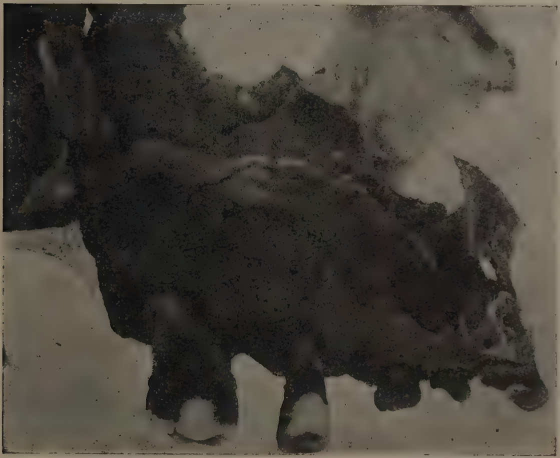

FIG. 524.—Side view of a specimen showing bone destruction in connection with a second incisor.

In the specimen (figs. 523 and 524) the pulp of the first incisor has been exposed by attrition, an abscess has formed around the apex of the root, and has then made its way through the outer alveolar plate. The second incisor, which shows marked attrition, has been attacked by caries and the suppuration of the pulp has been followed by infection of the bone. The destruction of the bone has laid bare the whole of the roots of the incisors and the upper aspect of the medial surface of the canine root. Extending backwards, the abscess has at one place completely destroyed the bone forming the palatal surface, so that one opening exists near the anterior palatine foramen. In its forward extension the abscess has opened into the cavity previously formed around the first incisor, and by this means there is a free communication through the outer alveolar plate. The radiogram of this specimen is seen in fig. 525.

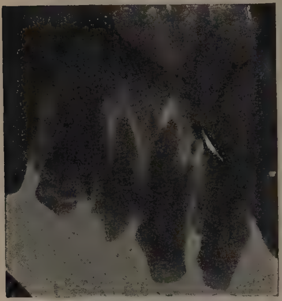

FIG. 525.—Radiogram of specimen shown in fig. 524.

This type of case would present the well-recognised clinical features of a palatal abscess. There would be a swelling in the palate, with a bulging of the tissues over the anterior aspect of the tooth and fluctuation between the two surfaces.

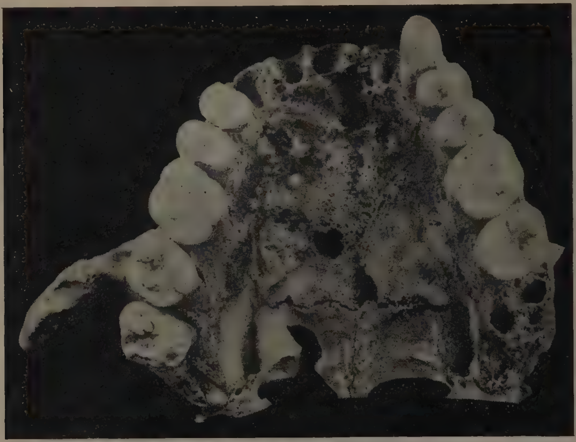

FIG. 526.—Palatal view of a dento-alveolar abscess encroaching on the nasal fossa.

The encroachment upon the nasal fossa of an alveolar abscess in connection with the incisors is shown in fig. 526. The teeth were all present at the time of death with the exception of the left first incisor. Viewed from the palatal aspect there is a slight elevation of the bone on the right side, the elevation extending from the anterior teeth to the suture between the maxilla and the palate bone. About 6 mm. from this suture and close to the median line there is an opening communicating with a large abscess cavity. The margins of the opening are irregular and the surrounding bone thin and porous. Viewed from the nasal aspect it will be observed that the bone forming the floor of the left nasal fossa has been pushed upwards and partly destroyed, so that a clear view is obtained of the abscess cavity, which measures roughly 25 mm. by 15 mm. The sockets of the right incisors and canine open into the cavity.

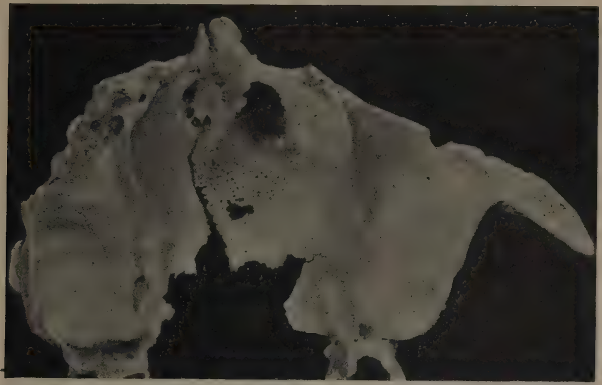

FIG. 527.—Nasal aspect of the maxilla shown in fig. 526.

The absence of the left first incisor was probably due to trauma and it is probable that the right incisors were injured at the same time, resulting in infection of the pulps. The abscess, as it increased in size, gradually destroyed the cancellous tissue and then, burrowing towards the root of the nasal process, gradually pushed up the bone forming the floor of the anterior nares and the internal aspect of the nasal process and completely blocked the anterior nares.

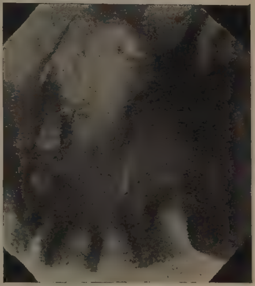

FIG. 528.—Radiogram of the specimen shown in figs. 526 and 527.

A radiogram of the specimen is shown in fig. 528.

An alveolar abscess in connection with the maxillary canine usually opens through the anterior alveolar plate about the region of the apex of the tooth. More rarely the pus will track towards the nasal fossa and, discharging into that cavity, give rise to a persistent unilateral nasal discharge. A specimen illustrating this latter condition is shown in fig. 529. Above the right canine there is a large excavation in the bone. The cavity is oval in shape and has been formed at the expense of the nasal process, the bone at one place being perforated so that the abscess cavity and the nasal fossa are connected.

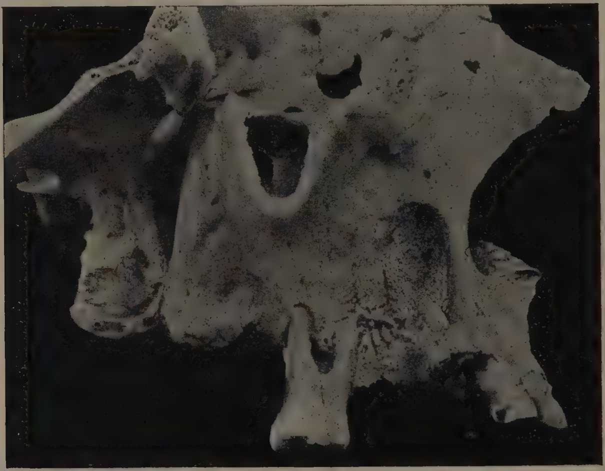

FIG. 529.—Specimen showing bone destruction in connection with the right canine. The abscess has opened into the nasal fossa.

The extensive destruction of bone that may occur around the roots of premolars is shown in figs. 530 and 531. The teeth have lost their bony surroundings except on the lower half of their palatal aspects, the destruction of the bone being especially noticeable in the region of the apices. If the teeth alone are removed healing is delayed, but if the bridge of bone holding them is also taken away free drainage is obtained and rapid healing of the wound follows.

In the mandible, the abscess is generally more restricted owing to the denser character of the bone, but very extensive excavation of the bone is occasionally seen. With the extension of suppuration, fresh bone is deposited by the periosteum, and, in this manner, the mandible may become considerably expanded.

The morbid anatomy of a case of chronic suppuration around a partly erupted mandibular third molar is shown in figs. 532 and 533. An examination of the specimen points to the condition having existed shortly before death, and it is possible that death

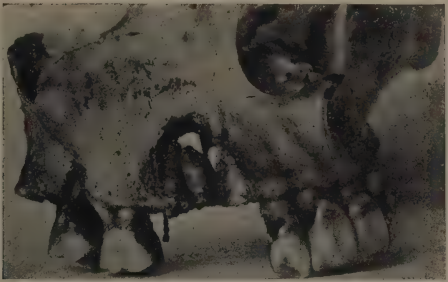

Fig. 530.—In this specimen there has been extensive bone destruction in connection with the premolars. View from the buccal aspect.

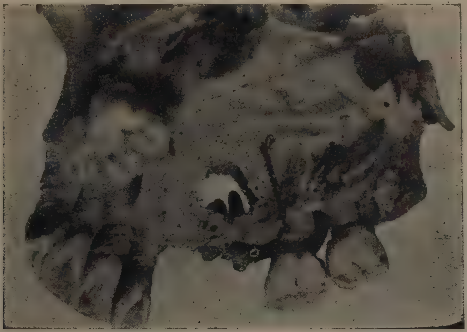

Fig. 531.—Palatal view of the specimen shown in fig. 530.

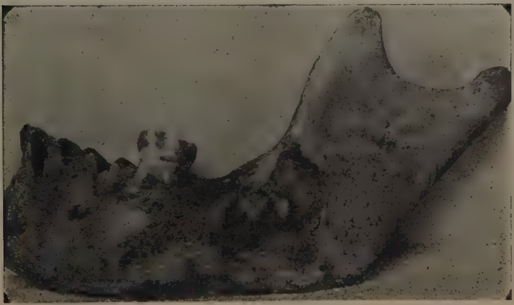

Fig. 532.—Mandible showing osteitis in connection with a mandibular molar.

was due to septicæmia from the mouth condition. A view of the inner aspect of the bone shows a deep cavity extending into the body of the bone to a point just below the level of the mandibular canal, which has been destroyed, leaving the nerve freely exposed. The external aspect of the bone shows a deposit of fresh bone which in the centre is pierced by an opening leading to the tooth socket. The radiogram (fig. 534) shows clearly the extent of the bone excavation and brings out the relation of the cavity to the mandibular canal.

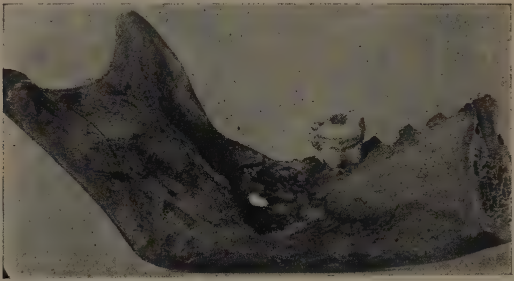

FIG. 533.—View of the internal aspect of the specimen in fig. 532, showing destruction of bone from suppuration around the third molar.

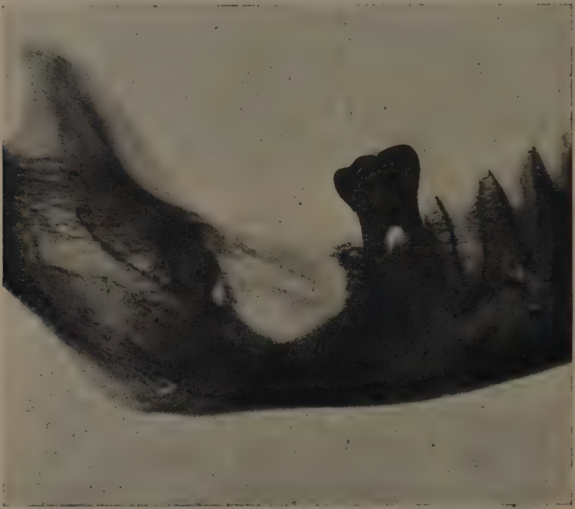

FIG. 534.—Radiogram of the specimen shown in fig. 533.

In the process of suppuration, the periodontal membrane may be destroyed, leading to necrosis of the cementum.

A somewhat rare form of abscess[1] which occurs in the jaws may be described under this heading, as it is in reality a sequel to suppurative periodontitis. The abscess may be caused by the presence of a small piece of root in conjunction with insufficient drainage after the extraction of septic teeth. J. G. Turner has drawn attention to this type of abscess. He describes the symptoms as follows: 'When the abscess occurs in the maxilla there is pain and tenderness, some swelling of soft parts, but no bony enlargement; the trouble may subside and occur again at intervals. If untreated the antrum may be involved. The nearest lymphatic glands may be enlarged. In the mandible, as in the maxilla, the alveolar process may be entirely absorbed, and there is pain and tenderness and some local swelling. Owing to the position of the inferior dental nerve there is often, especially in the premolar region, severe neuralgia. As in the maxilla, the nearest lymphatic gland may be enlarged and tender. The neuralgia is caused by the abscess, in its enlargement, opening up the canal of the nerve. Under these conditions there is very severe, continuous, heavy pain, and the more acute shooting pain which occurs at intervals without any necessary regularity; it is set up by any irritation, such as cold air, movement of eating, etc.; it spreads in anatomical order to the other branches of the fifth nerve, first to the ear and then up the side of the ear to the vertex, to the infra-orbital nerve, to the supra-orbital nerve, and down the neck. It lasts a varying time, from a few seconds to hours. It is unaccompanied by trophic changes, but there may be increased secretion of tears or saliva. It is confined to the one side, and appears always to start from the seat of mischief. If the abscess has not involved the inferior dental nerve, the neuralgia will be absent.'

'The length of time during which things may lie dormant varies greatly; when, therefore, a patient complains of pain or neuralgia starting in a spot where there is now no tooth, there may yet be a chronic abscess, and the alveolar process may be normally absorbed.'

Signs and Symptoms.—Where the injury is slight and leads to a *proliferative periodontitis*, symptoms are often absent and, when present, seldom amount to more than a slight tenderness on percussion and, occasionally, attacks of pain of a dull, gnawing character. *When the inflammatory process is of a rarefying type*, the principal symptom will be slight pain and tenderness on percussion, and, if the inflammation is near the apex and the pulp is alive, there will usually be signs of inflammation in it through extension of the disease to the pulp. The gum covering the oth may be swollen. As the tooth substance disappears, creased looseness will be noticeable, the tooth becoming more tender and the pulp and the gum more congested. In cases of doubt a radiogram should be obtained.

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[1] *Brit. Dent. Journ.*, 1899, 551.

A chronic suppurative periodontitis accompanied by a sinus will exhibit very few symptoms. Frequently the tooth causes no pain and the patient complains only of the presence of the ‘ gumboil ’ which periodically swells and bursts. The sinus is usually situated over the root of the tooth. In cases of sinus on the face, some little difficulty may be experienced in tracing the offending tooth, especially if two or three teeth are pulpless or septic. A digital examination of the sulcus will often disclose a fibrous-like cord running from the base of the root to the cheek. The root or tooth causing the trouble may be buried in the gum. A sinus in the mouth in connection with suppurative periodontitis must be diagnosed from sinuses connected with necrosed bone or foreign bodies. In cases of doubt, a radiogram will prove useful. When the sinus is on the face it should be diagnosed not only from cases due to necrosed bone or foreign bodies, but also from salivary fistula.

When a sinus is not present (*chronic alveolar abscess*) the tooth is usually slightly tender to percussion, the amount of pain varying according to the activity of the inflammation. The gum over the root of the tooth is at times swollen and congested, but may be quite normal. A swelling can, as a rule, be felt well up over the apex of the tooth ; this swelling is usually sensitive. In cases in which the maxilla or mandible is much involved, there is often little or no pain or tenderness. A chronic alveolar abscess must be diagnosed from dental cysts and other fluid swellings of the jaws (see Chapter XXXIII).

Treatment.—The treatment of chronic periodontitis must depend upon the cause. Where the pulp is living and the condition is traceable to excessive use (traumatic occlusion), relief can be obtained by ‘ freeing the bite,’ and applying counter-irritants to the gum.

When the chronic periodontitis is traceable to infection from the pulp canal the first question which demands attention is whether the retention of the tooth is advisable. A radiogram should be obtained and, if there are no signs of rarefying osteitis, an endeavour may be made to save the tooth by sterilising the tooth tissue through the pulp canal ; if this is not feasible the tooth should be removed. In the case of septic teeth associated with rarefying osteitis, thorough sterilisation of the canals will often be followed by the closing of a sinus, if it exists, and the subsidence of symptoms. Such teeth, although apparently cured, frequently remain a source of infection which may easily be overlooked, and extraction of the tooth is the safer line of treatment (see Chapter XIV). Knowledge acquired in recent years has emphasised the dangers of dental sepsis, and as a consequence our views as to treatment have been modified in the direction of removing septic teeth which we should formerly have endeavoured to retain. Sinuses in the mouth connected with septic teeth usually heal rapidly when the teeth are removed. Sinuses on the face or neck are sometimes slow in healing, and it may be necessary to stimulate the track with 10 per cent. solution of nitrate of silver, or a drug possessing a similar action. If this treatment fails, an anæsthetic should be given and the surface of the track scraped. Where a cavity exists in the bone over the tooth, the sinus should be opened up and the cavity scraped and packed to assist healing. Difficulty is sometimes experienced in the healing of palatal abscesses, and when this is the case a free excision of the muco-periosteum which covers them will often prove to be a satisfactory procedure.

It must be remembered that a local suppurative process may cause a local necrosis of bone, and that after the causative tooth has been removed a sequestrum may remain which is too large to find exit through whatever openings may be present ; such occasionally occurs in palatal abscesses, and suppuration will continue till the tissues are opened up sufficiently for the removal of the sequestrum after it has become separated from the live bone.

A depressed scar on the face may result from the healing of a sinus. In order to lessen the deformity, the attachment of the scar to the bone should be freed and a plate inserted until a sulcus is established. Should further treatment be desirable, the scar should be excised and the edges of the wound sutured with horse-hair ligatures.

Actinomycosis, Nocardiosis, or Streptothrix Infection.—Streptothrix infection in connection with the lesions of the teeth may be conveniently dealt with under 'Chronic Local Periodontitis,' inasmuch as the infection often takes place via the periodontal membrane.

Streptothrix in connection with lesions of the teeth may involve the bone itself or the soft parts, the latter condition being more usually met with.

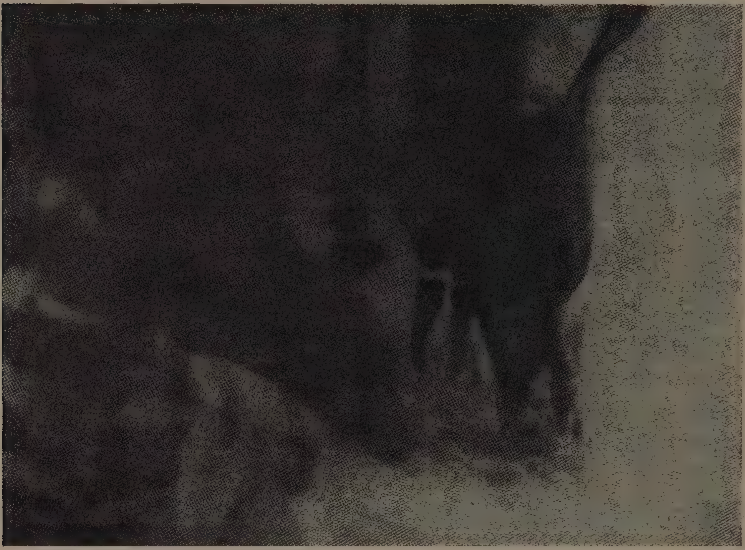

FIG. 535.

Clinical Features.—When the soft parts are involved a swelling appears, and gradually increases in size. At first the swelling has a regular and even surface, with the skin slightly hyperæmic. As the disease progresses, the tissue in places softens and nodular excrescences appear of a yellowish colour; these burst, giving exit to a semi-glutinous fluid in which the characteristic nodules of actinomycosis can be seen. The nodules vary in colour, being yellowish, yellowish-white, or quite clear. The wound generally heals and is followed by a fresh breaking out at another spot. The cicatrisation following the repetition of this phenomenon gives the surface a curious puckered appearance which is characteristic of this disease. If left untreated the disease spreads to the tissues of the neck, and in some cases passes upwards to the temporal region. The sinuses formed by the breaking down of the tissue may persist and a probe passed down them will often show that they lead to bare but not necrosed bone. The swelling is of a hard, brawny nature, and fades away gradually into the unaffected tissue. The disease is not accompanied by a rise in temperature, and there is usually no pain. Should suppuration supervene, the general symptoms of an ordinary abscess appear.

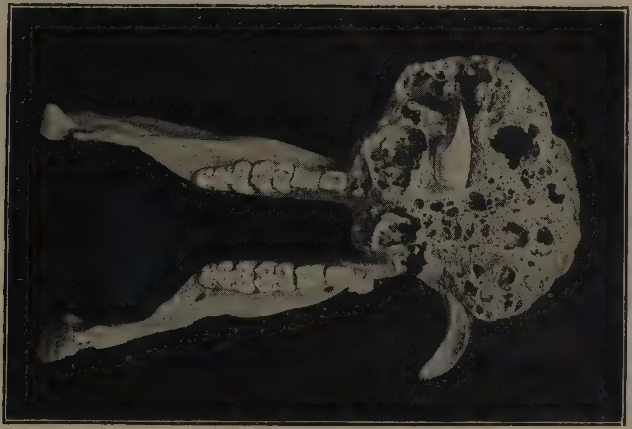

Fig. 536.

The lymph-glands are not enlarged unless secondary infection occurs.

The clinical features of streptothrix infection of the bone simulate necrosis. In the majority of cases there is a history of an acute onset of symptoms; the gums become swollen and bleed readily; the teeth loosen; and there is considerable swelling of the tissues covering the body of the bone. With the progress of the disease the infection spreads along the medullary cavity, clinically expressed by a well-defined edge to the swelling. Eventually the teeth are lost and portions of the bone exfoliate. Radiograms show the characteristic appearance as seen in fig. 535. An idea of the morbid anatomy of the condition can be gleaned from the mandible of a wild boar affected with streptothrix infection shown in fig. 536.

Diagnosis.—Streptothrix infection can be differentiated from alveolar abscess by the hard, brawny character of the swelling ; the long duration of the disease compared with that of alveolar abscess, and by the presence of the multiple sinuses or the puckered cicatrix, the latter being almost pathognomonic. From tubercle it may be differentiated by the fact that in tubercle the glands are usually affected, and the swelling is generally well defined and movable. From necrosis of the jaw it may be diagnosed by the presence of bare and not necrosed bone. The presence of granules in the discharge and the demonstration of the streptothrix on microscopical examination are positive evidence.

Pathology.—The disease is due to the presence of a form of streptothrix in the tissues, but the ray fungus of Bollinger (*Streptothrix bovis communis*) is an uncommon cause of human infection. The lesion produced by the growth of the streptothrix is of a chronic inflammatory character. A section through the affected tissue shows a number of rounded areas, in the centre of which is the streptothrix surrounded by a zone of granulation tissue. The tissue breaks down, and the streptothrix is expelled in the discharge. It is becoming more and more apparent that the relation of streptothricæ to disease is of considerable importance. A. G. Foulerton [1] has identified several species in man.

Lord, and Colebrook, have found actinomyces both in carious teeth and in tonsillar crypts ; the latter found it present in all of six carious teeth examined. A disease of a similar nature is very common among marsupials of the genus macropus.

Bjerrum and Hansen [2] (Denmark), examining the pus from 112 seemingly innocent chronic dental abscesses, were able to cultivate anærobic actinomyces from twenty-seven, but healing after the extraction of the tooth was complete and uneventful in every case. From thirty periodontitic teeth anærobic actinomyces were found on twenty.

Method of Infection.—The infection used to be thought to be conveyed by straw, cereals, etc., but this is not much credited now, as the organism is believed to occur commonly in the mouth ;

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[1] *Lancet*, 1910, March, 626.
See also :—
Foulerton, A. G. R. *Lancet*, 1913, February, 381.
Cope, V. Zachary. *Brit. Dent. Journ.*, xli, 649.

[2] *Ugeschrift for Laeger*, 1932, 1075.

one has seen cases in people who have spent their lives in thickly populated areas (Whitechapel, East Ham, etc.), who have never had anything to do with horses or rural life. The parasite probably gains an entrance to the tissue via the periodontal membrane, and thence spreads to the neighbouring soft tissue. In specimens of actinomycosis in oxen the invasion of the tissue via the periodontal membrane can often be demonstrated.

Naelsund [1] (Sweden) looks on the causative organism as occurring commonly with other thread-forming organisms in the mouth and forming a base for the deposition of salivary calculus, and only giving rise to signs and symptoms when circumstances, such as a breach of surface, are favourable for the organism. The following two cases suggest that the mouth may be the source of infection : Israel reported a case where a fragment of carious tooth was embedded in an actinomycotic focus in the lung ; and Cope a case of actinomycosis of the knuckles damaged by a blow which broke an antagonist's tooth.

From the above it would appear that the causative organism is often harboured in the apparently normal mouth.

Treatment.—Locally the softened spots must be incised and freely scraped and the sinus packed with iodoform gauze. Internally, iodide of potassium must be administered in doses increasing up to 50 or 60 gr. three times a day. It is also useful to add three to four minims of liquor arsenicalis, as this assists in preventing the skin lesions ; it is also considered by Kellock [2] to have some effect on the disease itself.

Tincture of iodine (without potassium iodide) in five minim doses three times a day is also used and occasionally collosal copper.

More recently X-rays have been used in conjunction with potassium iodide with good results ; it is believed that the X-rays help by increasing vascularity and promoting fibrosis, and possibly by being inimical to the streptothrix.

When the bone is involved, the teeth implicated in the affected area must be removed and free drainage established, combined with the administration of potassium iodide. Recovery is usually slow.

The prognosis is good, but the treatment may have to be carried out continuously for a long period.

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[1] *Proc. Roy. Soc. Med.*, 1930 (Sect. of Comparative Medicine).

[2] *Trans. Odonto. Soc.*, 1904, xxxvii, 29.

(ii) Commencing at the Gingival Margin.

When commencing at the gingival margin, chronic local periodontitis is usually due to trauma, such as the injudicious use of ligatures, elastic bands or mechanical contrivances in orthodontic treatment ; the too rapid separation of teeth for filling ; or the collection of food debris in the approximal space. As this classification implies, it is always accompanied by a marginal, and sometimes a more extensive, gingivitis.

Sometimes, apparently, it is due to remnants of the deciduous teeth remaining in the jaws for many years and so causing chronic irritation, and sometimes to the forcing of food, during the act of mastication, into the interproximal space between two teeth ; this is particularly the case when interstitial cavities are present.

Morbid Anatomy and Pathology.—One must always regard periodontitis of this variety as being a potential or probable forerunner of damage to more than the periodontal membrane, as it so constantly progresses as to involve the gingivæ generally and bone, *i.e.* though the disease commences as a marginal gingivitis or periodontitis it is very apt, by involving other structures, to become a parodontitis. The whole of the periodontal membrane of the tooth may be attacked. This occurs when the irritant is a retained ligature or an elastic band. The gum around the tooth is swollen, congested, and painful and the inflammation spreads to the periodontal membrane. By a process of epithelial invasion, the membrane is gradually destroyed, the root laid bare, and in time the apical vessels are involved, leading to pulp complications. The disease may be partial, *i.e.* the periodontal membrane on one side of the tooth only may be attacked. This condition is seen in the mandibular incisors on the labial aspects, where the membrane is occasionally destroyed to the apex of the root. The most important form from a clinical point of view is that met with in the region of the premolars and molars. A space may exist between the posterior teeth, with the result that food becomes wedged between the teeth during mastication. The lateral pressure thus caused starts chronic periodontal inflammation which, in time, leads to the destruction of the tissue, and the condition, if not treated, will gradually involve the membrane up to the apex of the root. In other cases, the teeth may approximate at the occluding surfaces, but, owing to a slight recession of the gum, food works its way between the approximal spaces during mastication. Unless these particles of food are completely removed a progressive ulceration of the periodontal membrane is started. The membrane is destroyed in a cup-shaped manner, as seen in the diagram (fig. 537), forming pockets, the gum apparently filling up the approximal spaces.

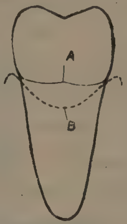

FIG. 537.—(A) neck of tooth and normal attachment of gum; (B) margin of gum.

The destruction of the membrane proceeds until the apical vessels are involved and pathological processes are then started in the pulp.

In practice a curious condition is met with in which the internal aspect of the palatine root of a maxillary molar is denuded of its socket. An examination of the root will frequently demonstrate a mass of calculus around the apex of the offending root in addition to a well-marked space between the tooth and its anterior neighbour. The pathology of this condition is not certain. An early stage is shown in fig. 538. In this specimen

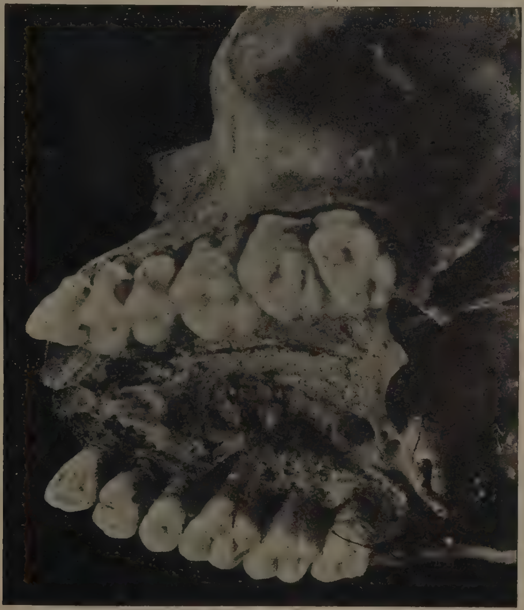

FIG. 538.—A specimen showing destruction of bone around the palatine root of the right first molar. (From *Dental Record.*)

the jaw is well developed, the teeth are free from caries and are arranged in a good arch ; there is a slight general destruction of the alveolar process, a deposit of dark calculus around the necks of the teeth on the palatine aspects and a fair deposit of lighter coloured calculus on the buccal aspects of all the teeth. In the region between the right second premolar and the first molar there is a cavity in the bone ; the tissue that has disappeared comprises the septum between the teeth and the anterior and internal aspects of the socket of the palatine root.

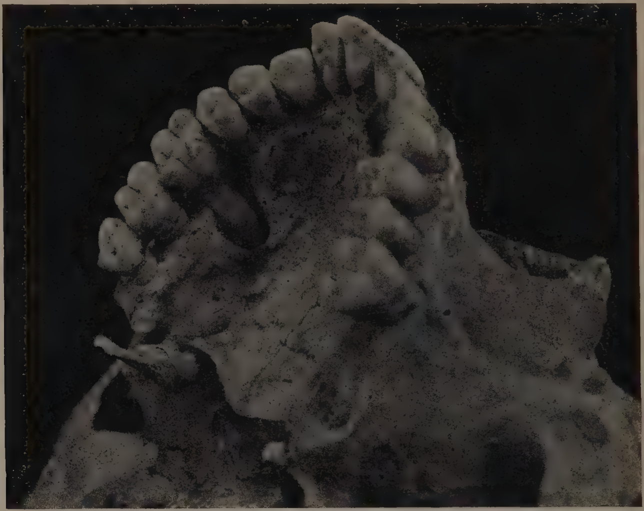

Fig. 539.—A specimen showing extensive destruction of bone around the palatine root of a maxillary right first molar. (From *Dental Record.*)

On the apical portion of this root there is an irregular deposit of hard dark calculus. The surface of the cavity is regular and smooth, suggesting that the cavity is the result of a slow, but constant, excavation.

A more advanced condition is illustrated in fig. 539. Here the process of excavation has extended as as to expose almost the entire palatine root. The edge of the cavity is smooth and regular, indicating, as in the previous specimen, that the destructive action has been slow, but constant.

In both the specimens the destruction of bone appears to have commenced in the interval between the second premolar and the first molar, and this would suggest that the primary cause of the lesion was probably a retained portion of a deciduous tooth, which had formed a stagnation area, the infection gradually involving the adjacent teeth.

Inflammatory processes around the neck of the tooth occasionally cause rapid destruction of the tooth tissue and the formation of definite cavities. The teeth shown in fig. 540 were removed from a young man who had been injured in an air crash. The canine and first premolar had been driven into the bone and only the occluding surfaces of the teeth were showing. They had been in this position for nearly six weeks. On the palatine aspect of the premolar there was a cavity which had spread slightly upwards, and under the enamel; examination with a lens showed that it was due to absorption of the tissues. A similar but smaller cavity was found on the canine on the distal aspect. A case in which the absorption was extensive is shown in fig. 541. Here, in a mandibular canine, the tooth tissue with the exception of that around the pulp canal had been lost by the process of absorption. There is no doubt that some of the cavities around the cervical region are produced in this way and are not the result of caries. The pathology of the condition is not clear, but it is reasonable to think that the condition is due to inflammatory reaction in the periodontal membrane.

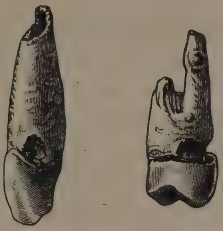

Fig. 540.

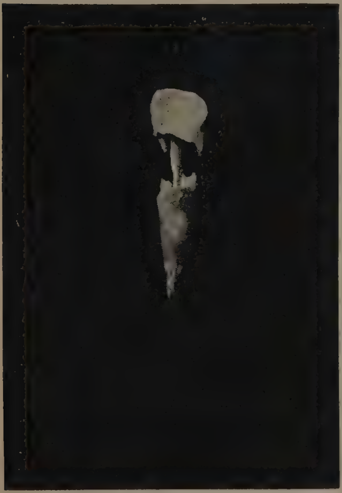

Fig. 541.
(From *Trans. Odonto. Soc.*)

Signs and Symptoms.—Slight tenderness during mastication, with pulp pain from thermal changes (owing to exposure of the cementum), are the chief symptoms. *In cases due to ligatures or elastic bands,* the gum shows signs of inflammation and pus can usually be squeezed up round the neck of the tooth. There is pain to thermal changes. *In cases due to pressure from mastication* there is extreme tenderness, the gum is swollen and congested and the tooth is tender to pressure.

When a pocket exists and the occluding surfaces are in contact, the tags of gum on the labial and palatal aspects of the approximal space appear slightly congested, but the recession from the space is, in many cases, only slightly marked. The patient complains of slight discomfort from food becoming wedged between the teeth and, if there is pain to thermal changes, an examination with a suitable probe will show a considerable destruction of periodontal membrane and bone, the root of the tooth being frequently denuded to the extent of at least half its length.

When a pocket occurs between the maxillary molars there is often a tender area over the region of the malar bone.

The diagnosis presents little difficulty. An inflamed condition of the gum ; slight tenderness on percussion and pain on touching the exposed cement are sufficient evidence. Care must, however, be taken to exclude pulp irritation from other causes.

Treatment.—*In cases arising from ligatures, elastic bands, calculus, etc.*, the cause must necessarily be removed. The socket is then syringed with a suitable antiseptic (hydrogen peroxide, 2 vols.) and the ulcerated surface treated with some mild escharotic, such as trichloracetic acid (25 per cent. solution).

In cases due to pressure from mastication, careful contour fillings must be made in the approximal surfaces of the teeth and the exposed cementum treated with nitrate of silver. When the periodontitis is *traceable to the lodgment of food solely*, and is not due to pressure from mastication, the approximal space should be syringed and the surface of the gum cauterised with nitrate of silver. This drug will serve a double purpose, namely, reduce the sensitiveness of the cementum and at the same time promote the formation of healthy granulations in the approximal space. The application by the patient of tincture of iodine every day for a week will result in the disappearance of the pocket and the formation of a space which can be easily kept free from food debris.

(3) General Periodontitis.

Acute.—In this condition the morbid process involves several or many teeth as well as the adjacent periosteum of bone. A general inflammation of the whole alveolar periosteum has therefore to be dealt with. The condition may have its origin in the periodontal membrane or in the muco-periosteum. The teeth become loose and sensitive to touch. The muco-periosteum is swollen, deeply congested and painful to pressure. The inflammation may resolve, but suppuration is very likely to occur unless prompt measures are taken to relieve the congestion. If suppuration occurs, the periosteum becomes separated from the bone, with the result that necrosis supervenes. The inflammation occasionally passes into a chronic form.

Causes.—The causes of acute general periodontitis are the same as those which may cause necrosis of the jaws (see Chapter XXX); as to whether the condition goes on to cause necrosis depends on the intensity with which those causes act and the resistance of the individual. Such causes are: exposure to the fumes of phosphorus, overdosage with drugs such as mercury or bismuth, and infection with syphilis. Very slight trauma during the lowered resistance of acute specific fevers may also cause it and, in addition, it occurs during scurvy, and as a result of severe trauma injuring several teeth and in acute exacerbations during the course of parodontal disease.

Signs and Symptoms.—The local signs and symptoms are similar to those of the local form, except that several teeth are involved in the process. Pain of a dull, gnawing character is present, and there will be general symptoms of pyrexia. Suppuration, if it occurs, is usually ushered in by a well-marked rigor, and the local pain becomes of a throbbing character.

Treatment. *Local.*—The application of fomentations with incisions for drainage if necessary. The mouth should be kept as clean as possible.

The *general* treatment will depend upon the cause. The bowels should be kept open in all cases. If the inflammation shows a tendency to suppurate, free incisions must be made down to the bone, followed by the constant use of fomentations. Cases due to a specific cause call for the general treatment of that condition.

Chronic.—Chronic periodontitis occurs in people of a gouty or rheumatic diathesis, in people suffering from malaria or syphilis, and after influenza; in addition, there is reason to believe that the periodontal membrane may be affected by toxins circulating in the blood-stream. Occasionally there is a thickening of the cementum on the teeth with no evidence of infection from the gingival margin. The following case illustrates this condition: A female aged thirty-five complained of indefinite pains about the head and a general feeling of ill-health. There was a history of nasal trouble. The gums were perfectly normal and there were no signs of infection around the necks of the teeth. On percussion several of the teeth were tender. Radiograms showed a definite thickening of the roots of the teeth, more especially the premolars and molars. Several of the teeth were removed with no relief to the general symptoms. The nasal condition was treated and relieved. Signs of chronic arthritis subsequently developed and further radiograms showed an increase in the cementum of the remaining teeth. The chronic periodontitis would here seem to be due to a cause acting through the blood-stream.

In other cases presenting little or no gingivitis and only vague clinical symptoms radiograms may show that absorption of the roots of the teeth is going on in the majority or all of the teeth, actively in some and intermittently in others ; no local cause can be found for the condition, it may be one acting through the blood-stream.

Signs and Symptoms.—Except that they are more generalised these are similar to those which occur when the affection is local.

Treatment.—This is dependent on the cause : if due to syphilis, antisyphilitic treatment is indicated, while cases of gouty or rheumatic origin call for the general treatment of those affections. Regularity of the bowels should be attended to and tonics administered as required.

(B) REGRESSIVE CHANGES.

The periodontal membrane of elderly persons is at times subject to regressive changes of a fibroid character,[1] which may also occur in persons of less mature years. The normal elements of the parts are replaced by fibrous tissue which is grouped together in bundles enclosing areolar spaces (fig. 542). The adjacent bone shows signs of atrophy and the gingival tissue in the immediate neighbourhood also shares in the changes by becoming less vascular and more fibroid in character.

(C) NECROSIS OF THE TEETH.

A tooth derives its nutritional supply from two sources, namely, the pulp and the periodontal membrane. When the death of the pulp alone occurs, *necrosis of the dentine takes place.* From a clinical point of view, this is unimportant and does not give rise to any symptoms if the canal is properly treated. When, in addition to death of the pulp, the periodontal membrane becomes separated from the cementum, partial or complete necrosis of the tooth takes place. *Partial necrosis of the cementum* is common and is generally the result of suppurative periodontitis. When there is *complete necrosis of the cementum*, the tooth becomes a foreign body.

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[1] A. Hopewell-Smith, *Dental Cosmos*, xlvi, 261.

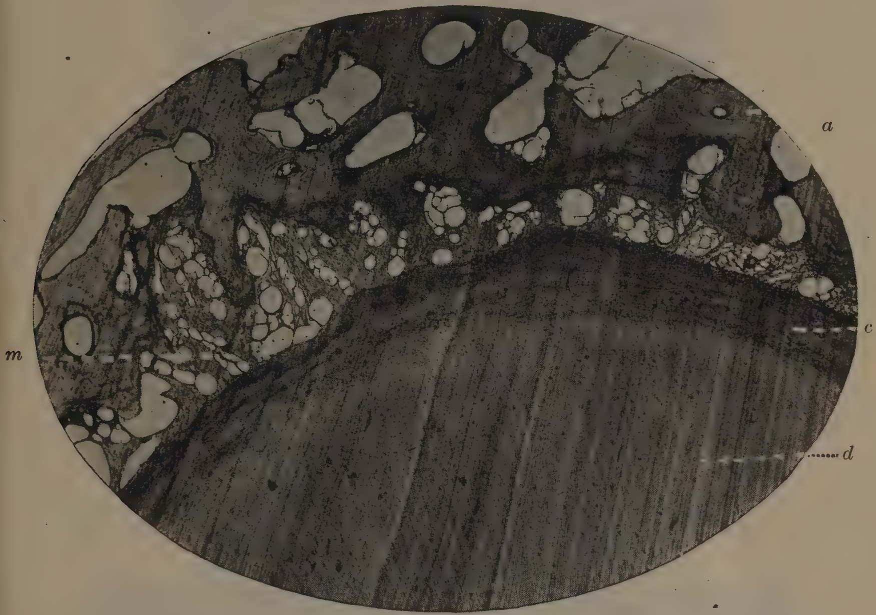

Fig. 542.—Transverse section of fibrosis of the periodontal membrane, *in situ.* × 50. (*d*) dentine ; (*c*) cementum ; (*a*) alveolar bone ; (*m*) fibroid periodontal membrane ; devoid of cellular elements and vascular and nervous systems. (A. Hopewell-Smith.) (From *Dental Cosmos.*)

(D) GRANULOMATA.

This is the term used to describe those masses of tissue often seen in connection with teeth which have been the seat of chronic periodontitis. They occur commonly at the apices of permanent teeth (fig. 543) which are pulpless and in which the infection has travelled through the apical foramina into the periapical tissues.

Occasionally they are found having origin from the lateral aspect of a tooth (fig. 544); when this is so, infection has sometimes stimulated their growth there by finding its exit through some aberrant opening connecting the pulp canal with the periodontal membrane (see fig. 545). They occur also on teeth with live pulps and are then apparently caused by irritation derived from infection in a parodontal pocket which gives rise to

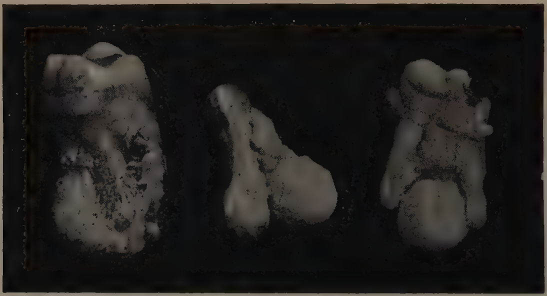

FIG. 543.

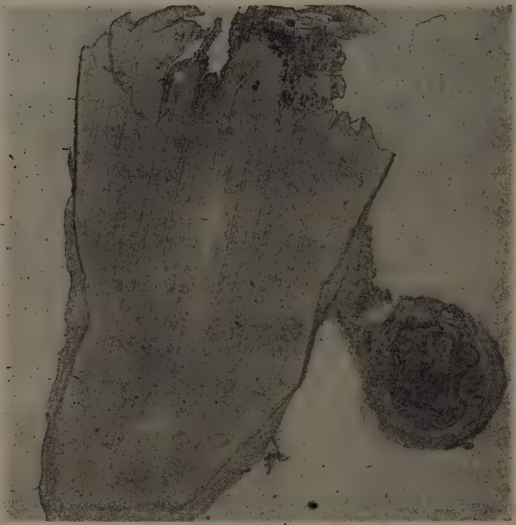

FIG. 544.—Granuloma on lateral aspect of a tooth. (E. S.)

them in a manner similar to when they occur at the apices of teeth.

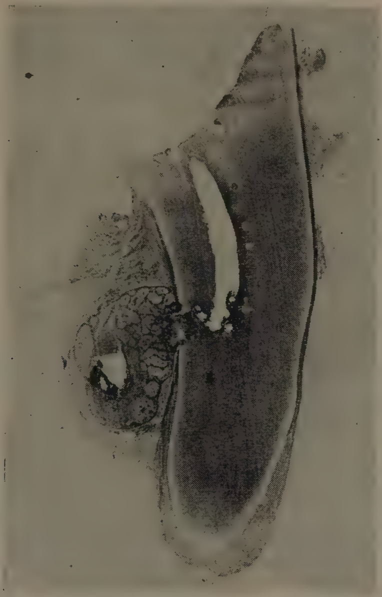

Fig. 545.—Granuloma with much epithelial proliferation and becoming cystic in centre formed at end of aberrant (pathological) opening into the pulp canal. (E. S.) (From *Brit. Dent. Journ.*)

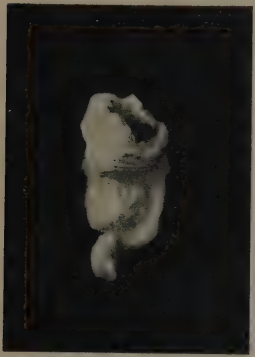

Fig. 546.—Granuloma on deciduous tooth. (E. S.)

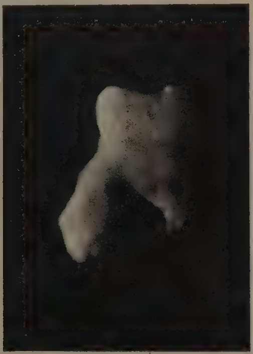

Fig. 547.—Granuloma on deciduous tooth. (E. S.)

(Now in Odontological collection of the Royal College of Surgeons of England.) (From *Proc. Roy. Soc. Med.*)

They also occur on deciduous teeth (figs. 546 and 547); they were first recorded on such teeth in 1922,[1] when one showed sections of them, and again in 1927,[1] when one showed many other sections and specimens ; their appearance on the deciduous teeth is rare because the short time that these teeth are present in the mouth does not give much time for their formation, and, moreover, as the roots of the deciduous teeth become absorbed they may become separated from their source of origin and so pass unnoticed when the teeth are extracted.

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), 1922, 15, 56. (E. S.)

Structure.—In structure and nature they nearly all consist of masses of infected granulation tissue with more or less epithelial proliferation, and vary according to their age and according to the age of the individual ; but rarely the mass is found to consist simply of a chronic abscess sac and have no epithelial proliferation (fig. 548).

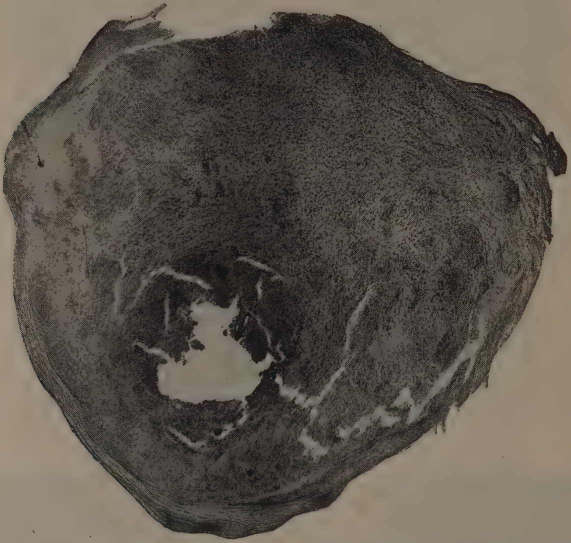

FIG. 548.—Small chronic abscess sac from apex of a pulpless tooth. There is no epithelial proliferation in it. (E. S.)

A recently formed granuloma usually consists of a mass of granulation tissue with a considerable amount of infection present, and an enormous number of inflammatory (plasma) cells infiltrating it (fig. 549). Ramifying throughout its structure are columns and processes of epithelial cells also infiltrated with inflammatory cells, and these latter often render the detection of the epithelial elements difficult.

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.). 1927. 20. 67. (E. S.)

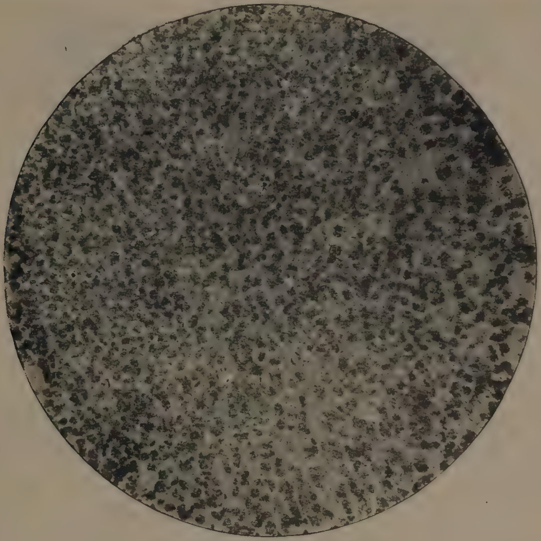

FIG. 549.—A granuloma of the periodontal membrane. (From 'The Histology and Patho-histology of the Teeth,' A. Hopewell-Smith.)

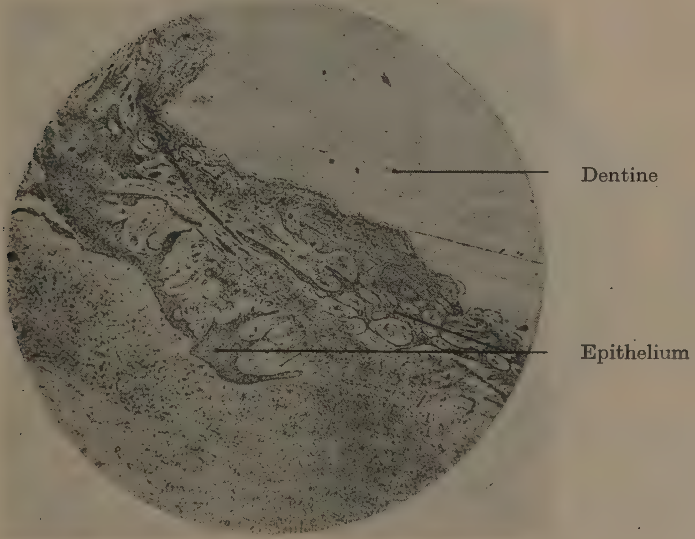

FIG. 550.—Photomicrograph of section of granuloma on deciduous tooth *in situ*. (E. S.) (From *Proc. Roy. Soc. Med.*)

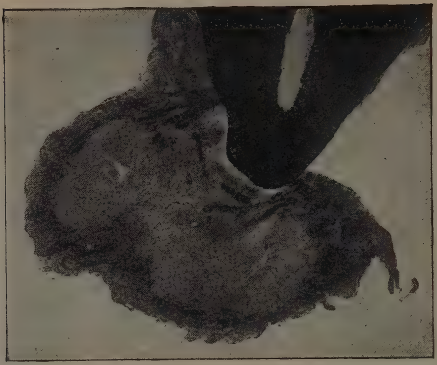

FIG. 551.—Photomicrograph of a granuloma stained with Mallory's phosphotungstic acid hæmatoxylin method to bring out the fibrous part of the tissue. Note the strong fibrous capsule. The inner part of the granuloma shows centres where necrosis has taken place. (Thoma.) (From *Dental Cosmos.*)

In all cases bone has been lost by a process of rarefying osteitis in order to accommodate the new mass, and often areas

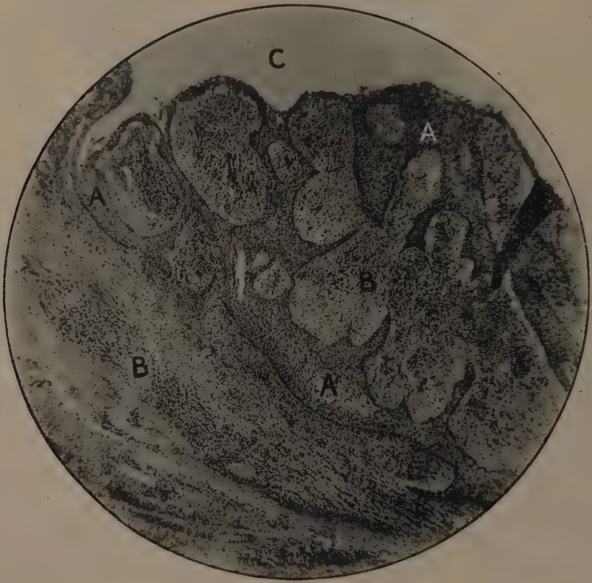

FIG. 552.—Section of part of an epithelial granuloma; C is placed in central cleft. A, trabeculæ of epithelial reticulum, central cells degenerating; B, mesoblastic tissue. (Turner.)

of degeneration or necrosis may be seen in the soft tissues (figs. 550, 551, and 552).

A granuloma of long standing is very similar, but presents a much more fibrosed matrix and on occasions a fibrous capsule.

In a granuloma on a deciduous tooth (*i.e.* from a young individual) the general structure is of the same nature as that of a recently formed granuloma, but the epithelial proliferation is usually more exuberant and ramifies over wider areas ; this is because at this age the jaws and teeth are actively growing and developing, the environment is more vascular, and the epithelial cells younger and therefore more vigorous. In a granuloma on a tooth of a person aged—say fifty, the epithelial growth in it is derived from 'rests' of Malassez which have been lying dormant and inactive for perhaps forty years ; one might therefore reasonably expect epithelial growth to be more exuberant in a granuloma on a deciduous tooth considering the youth of the cells of origin and the greater vascularity around them.

Occasionally what appears clinically as a granuloma is, on microscopical examination, shown to be the sac of a small chronic abscess and no epithelial cells may be found (fig. 548). Nearly all granulomata have some epithelial proliferation present in them.

In two specimens described by Romer, granulation tissue had found its way into the root canals, and only in that situation was he able to demonstrate epithelial remnants.

Areas of mucous tissue have also been described in granulomata. By some people, the fact that a granuloma is present is looked on as indicating that the tissues have reacted strongly in their attempt to localise and shut off the infection, and though this may be so it does not alter the fact that there is no escape for the products of bacterial growth from them save through the general circulation. The infecting organisms are usually streptococci, commonly the *Streptococcus viridans*, occasionally accompanied by staphylococci ; occasionally also hæmolytic streptococci may be present.

The epithelial granuloma is the first stage in the development of a dental cyst, and in many cases where granulomata are cut for microscopical examination small cysts are found to be present.

It is quite possible that some of the varieties of malignant neoplasms met with in the jaws originate in the periodontal membrane and the epithelium it contains. In a case recorded by

Oakley Coles[1] of a mandibular permanent second molar which had been the seat of chronic periodontitis, the structure of the small growth removed with the teeth was identical in character with a round-celled sarcoma. Hopewell-Smith[2] has also described cases of a similar nature.

(E) ANCHYLOSIS OF THE TEETH TO THE JAWS.

Anchylosis of the teeth to the jaws may occur, but it is rare. The pathological changes which produce this condition between the teeth and the alveolar process are similar to those occurring in joints in other parts of the body. An interesting case came under the notice of E. Lloyd-Williams. The patient was a man suffering from suppuration, and a maxillary molar and premolar were removed with large masses of bone attached to them. Examination of the specimens showed them to be examples of anchylosis of teeth to bone. The molar is shown in fig. 553, while a section of the premolar is seen in fig. 554. It will be noticed that the tooth and bone are intimately connected. An interesting case of anchylosis to the jaw of a retained deciduous molar in a man aged forty is described by O. Amoedo.[3]

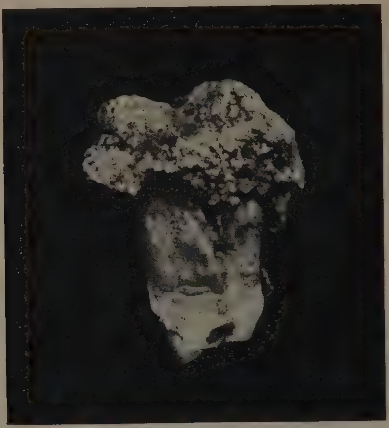

Fig. 553. (From *Trans. Odonto. Soc.*)

Anchylosis also occurs occasionally in teeth which remain unerupted in the jaws (see fig. 555); areas of the absorption of enamel, dentine, or cementum are not very rare on such teeth, and occasionally one finds that the lost tissue has been replaced by bone in direct connection with adjacent skeletal bone.

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[1] *Trans. Odonto. Soc.*, xi, 115.

[2] *Brit. Dent. Journ.*, xvi, 578.

[3] *Brit. Dent. Journ.*, xvi, 249.

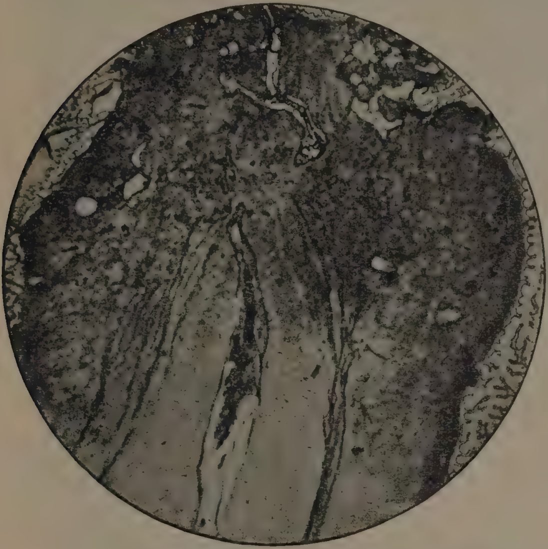

FIG. 554. (From *Trans. Odonto. Soc.*)

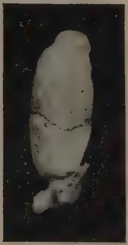

FIG. 555.—Unerupted maxillary canine from an adult, absorption of enamel has been followed by bony anchylosis. (London Hospital Dental Museum.) (E. S.)

More recently G. B. Pritchard[1] has investigated the anchylosis of teeth, and describes two conditions under which it may occur:

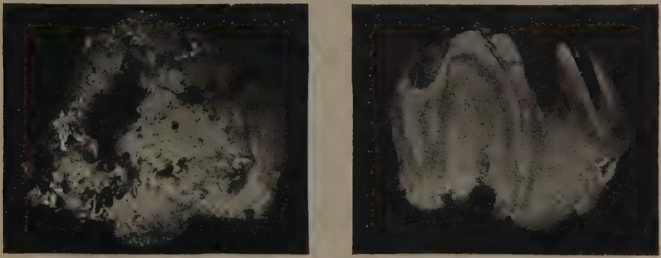

FIG. 556.—Anchylosis by Exostosis in mandibular left premolar and molar region, showing external and cut surfaces × 2. The external surface is rough; the cut surface shows a mass of bone obliterating the root bifurcation of the molar and uniting it to the premolar. (G. B. Pritchard.) (From *Brit. Dent. Journ.*)

both are associated with chronic infection. One he describes as *Anchylosis by Exostosis*; this occurs in pulpless teeth where apparently toxic irritation stimulates excess formation of cementum; there is a similar thickening of adjacent bone till the

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[1] *Brit. Dent. Journ.*, 1930, March 15; 1937, lxiii, 13.

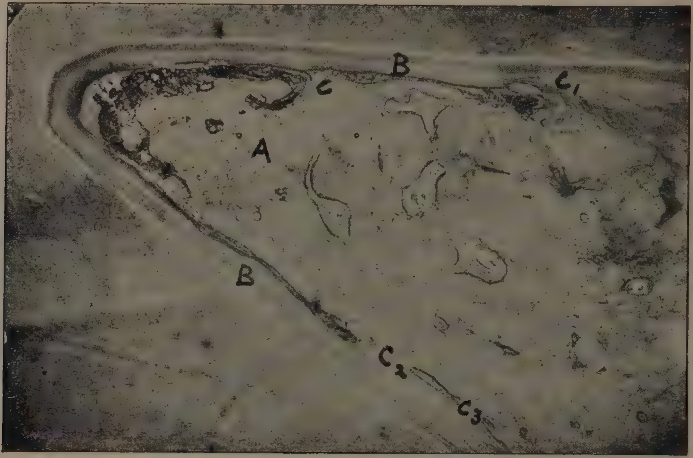

Fig. 557.—Section of molar root bifurcation shown in fig. 556 × 25, showing intermittent union of bone and cementum at C and C_2. The union from C_1 and C_3 to the apices is continuous. (G. B. Pritchard.) (From *Brit. Dent. Journ.*)

Fig. 558.—Anchylosis by Inostosis in unerupted mandibular right third molar, middle-aged male. (G. B. Pritchard.) (From *Brit. Dent. Journ.*)

two tissues meet and coalesce. The infection causes the separation of the bone at a distance from its tooth nucleus so that it dies and tends to be exfoliated, still in calcific union with the tooth (figs. 556 and 557). The second type, described as *Anchylosis by Inostosis*, seems to occur with unerupted or partly erupted teeth, chiefly mandibular third molars, in middle-aged people, and to be associated with the overlying gum flap or the follicle. Caries may or may not be present and is apparently irrelevant. Lacunar absorption takes place on the external aspect of the tooth of enamel, dentine or cementum or all of these tissues, followed by the production of new bone in continuity with them and the skeletal bone (figs. 558 and 559 and fig. 555).

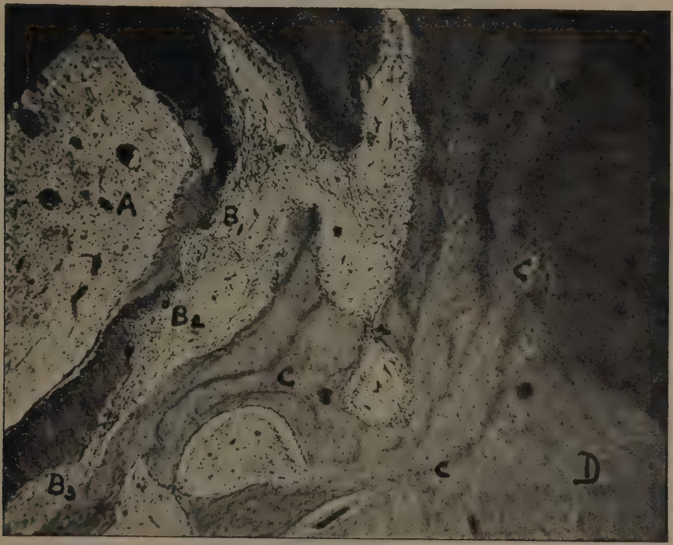

FIG. 559.—Section of molar shown in fig. 558 × 50. A, pulp; B_1, B_2, B_3, lacunar absorption approaching odontogenetic zone; C, bone; D, dentine of root. (G. B. Pritchard.) (From *Brit. Dent. Journ.*)

CHAPTER XVI

Diseases of the Gums and Adjacent Mucous Membrane.

General Hyperplasia of the Gums—Gingivitis—Stomatitis—Leucoplakia—Pemphigus—Purpura—Myelogenous Leukœmia—Scurvy—Perforating Ulcer—Tubercle—Syphilitic Lesions—Periarteritis Nodosa—Aneurism—Discolorations and Pigmentations—Neoplasms.

(A) GENERAL HYPERPLASIA OF THE GUMS.

This disease has heretofore been called 'hypertrophy of the gums.'

In its pathological sense *hypertrophy* means an increase in size due to the increased size of individual cells (but not to an increase in the number of cells), such, for instance, as is believed to occur in the uterus during pregnancy.

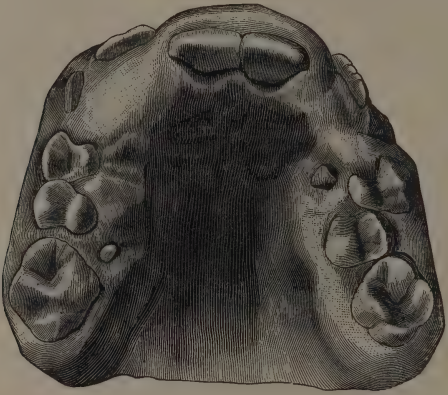

Fig. 560.—F. S., aged nine years (maxilla).

In the disease under consideration there is a considerable increase in the number of cells, chiefly fibroblasts, though the epithelial covering is, apart from its increased extent, normal.

The nomenclature of the disease therefore needs reconsideration and, as *hyperplasia* means 'overgrowth of a part due to a multiplication of its elements,' there can be no doubt that this term more correctly describes the condition.

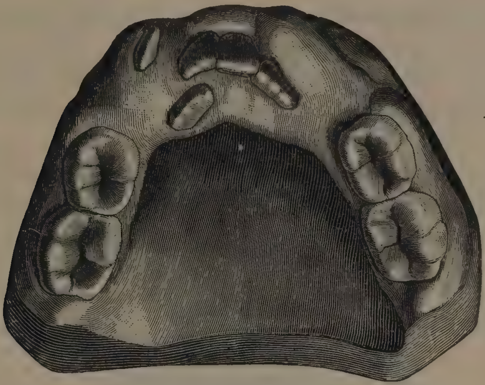
FIG. 561.—F. S., aged nine years (mandible).

Hyperplasia of the gums consists in a general overgrowth of that portion of the gums which covers the alveolar process. Typical examples of this disease are represented in figs. 560 to 563, which show the mouths of two brothers, aged seven and nine respectively. In a younger brother seen five years later at the age of six a similar condition was found, the thickening, according

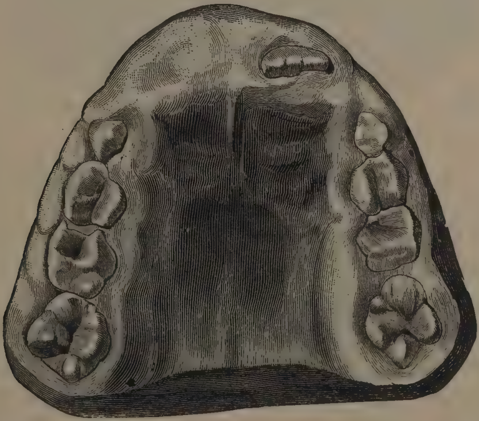
FIG. 562.—T. S., aged seven years (maxilla). (*Lancet*, 1897.)

to the father's statement, having begun as the teeth erupted. The hyperplasic gum in all three cases was normal in colour, of a dense fibrous character, and was more marked in the front of the mouth. The mouths were clean and there was no tendency to bleed and no discharge. The boys were intelligent and there were no lesions discoverable elsewhere. The parents and the two sisters were free from any abnormality of the gums.

The hyperplasia of the gums would seem to start with the eruption of the teeth. In a case quoted by Erichsen, the affection showed itself at the age of seven months, during the eruption of the incisors, and in another case, quoted by Heath, the swelling of the gums commenced at two years of age with the eruption of the deciduous molars.

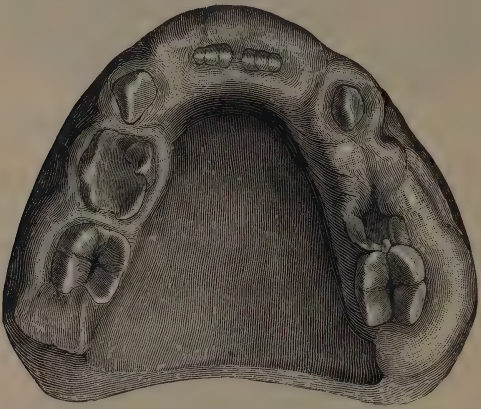

Fig. 563.—T. S., aged seven years (mandible).

In a case, recorded by D. Perkoff [1] and seen by one of us, which commenced in childhood, only a few of the permanent teeth had ever erupted, though radiograms showed that all were present except the mandibular third molars, which were congenitally absent; the disease had started in early childhood, there had been no history of eruption of deciduous teeth at all.

In his inquiry concerning this disease and its bibliography Perkoff produced some evidence that hyperpituitarism was very likely a causative factor, particularly in those recorded cases (six including his own) which showed excessive hairiness. It is known that the anterior lobe of the pituitary gland is concerned with the growth of the skeleton, skin, and soft tissues, and on embryological grounds there is the evidence that the anterior lobe is in close relation with the buccal cavity in that it is developed from the buccal epiblast as a tubular prolongation ; it soon, however, loses all connection with it.

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[1] *Lancet*, 1929, i, 1294.

If left untreated, the hyperplasia increases, and in time the teeth become completely hidden or their eruption may be prevented.

W. W. James has recorded [1] a case where hyperplasia of the fibrous tissue of the gingival muco-periosteum occurred with the eruption of each tooth of the deciduous and permanent dentitions.

In a case under the writer's observation for seven years a girl first showed the condition appearing at seven years on the left side of both maxilla and mandible, the gingival crest ultimately reaching the level of the molar occlusal surfaces ; in the course of a year or so the right side became similarly affected, and later the incisor regions. After the removal of the redundant gum there was no recurrence for four years, after which the patient was lost sight of. There was no excessive hairiness or other abnormality found in this case.

Mirolli [2] records two cases of marked hyperplasia of the gums, in a male aged 28 and his aunt aged 70 ; they were members of a family in which 12 out of 29 members were so affected in varying degrees. In none did the affection show itself till the second dentition. Mirolli was inclined to believe that some defect in the hypophysis was the probable cause, and this view was supported by the acromegalic appearance of the aunt.

On **microscopical examination** the structure is apparently that of a diffuse fibroma, but masses of inflammatory cells occur here and there, probably owing their origin partly to the trauma of mastication, and partly to the fact that the enormously increased depth of the gingival troughs creates much greater stagnation areas with corresponding increased irritation from their contents, which irritation, however, varies with the degree of infection present. The increase in size has also been thought to be brought about by lymphatic stasis following on infection.

A microscopical examination made by C. S. Tomes of one of Heath's cases showed that ' the growth closely resembled that of the small polypi which are sometimes found occupying the cavity of the carious teeth; it was true hypertrophy (as then called) of the gum, and chiefly of the fibrous portion. It sprang from the periosteum round the neck of the tooth just within the margin of the alveoli. From this point emanated a dense stroma of interlacing fibres, covered by a thin mucous and epithelial layer.'

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[1] *Lancet*, 1919, July 26.

[2] *Arch. Ital. di Chir.*, 1931, June, 401.

In many of the accounts given of the disease, the gums are described as being darker than the normal, and as bleeding freely at the lightest touch. This condition of the gums is due to inflammatory reaction in the hyperplasic tissue from sepsis in the spaces between the teeth and gums.

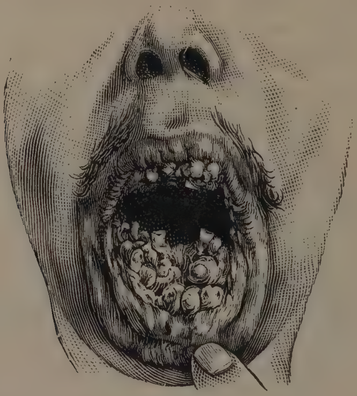

Fig. 564.

The combination of hyperplasia of the gums with superadded inflammatory reaction may give rise to a most distressing condition, of which the following case, recorded by Heath,[1] is a typical example. The patient was a man aged 26; four years before being seen he had consulted a surgeon, who excised the gum excess of both jaws and afforded considerable relief to the patient. Three months later the growth had recommenced, and, although it had made steady progress for three years, the patient sought no further advice. On admission to hospital, the external deformity was well marked, and, on opening the mouth, the gums of both jaws were seen to be enormously enlarged, most of the teeth being loose and displaced (fig. 564). The palate at a cursory glance had the appearance of a cleft palate, but this was due to the fact that the hyperplasic gum on either side covered the palate almost to the median line.

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[1] *Brit. Med. Journ.*, 1897, i, 1073. and *Lancet*, 1897.

A microscopical examination of the hyperplasic tissue showed that the mucous membrane covering the growth was healthy, the bulk of it being composed of delicate bundles of wavy fibrous tissue which interlaced. Between the bundles were numerous cells, in some places forming large clusters. Numerous vessels were scattered through the growth.

A case exhibiting another clinical variety of hyperplasia of the gums is shown in fig. 565. In this variety the surface of the gum is nodular and uneven instead of being smooth. This patient, a man aged 43, was under the care of Montagu Hopson.[1] Two of his children also showed slight hyperplasia of the gums.

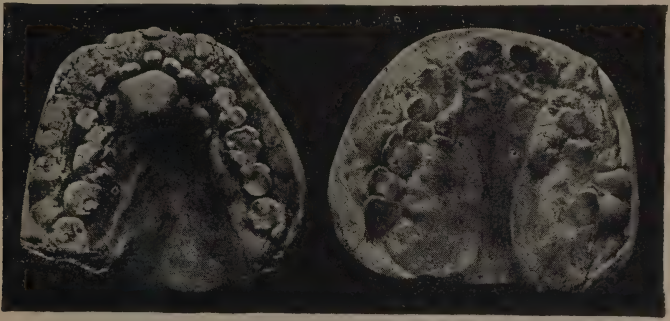

FIG. 565. (From *Trans. Odonto. Soc.*)

Hyperplasia of the gums is at times correlated with abnormalities of other tissues. In three cases recorded by Murray,[2] which occurred in three children (two girls and a boy), the condition was associated with molluscum fibrosum and defective mentality.

In the well-known case of Julia Pastrana (fig. 566) hyperplasia of the alveolar process and overlying soft tissues was accompanied by excessive development of the hair. A similar condition existed in a girl, Krao, from Laos in Burmah, aged seven years, mentioned by Parreidt.[3] The enlarged gums were accompanied by a well-developed growth of hair on the body. It is possible that some of the earlier cases of absence of teeth associated with excessive hairiness may have been of this nature.

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[1] *Trans. Odonto. Soc.*, xxxii, 36.

[2] *Med. Chir. Trans.*, lvi.

[3] *Deutsche Monatsschrift für Zahnheilkunde*, 1886, Jahrgang iv, H. 2.

Treatment.—Microscopical sections demonstrate that the hyperplasic gum springs from the alveolar bone. In treatment, therefore, it may be necessary to remove the affected alveolar process as well as the excess of gum, as there is a possibility that the trouble may recur if the gum only is removed.

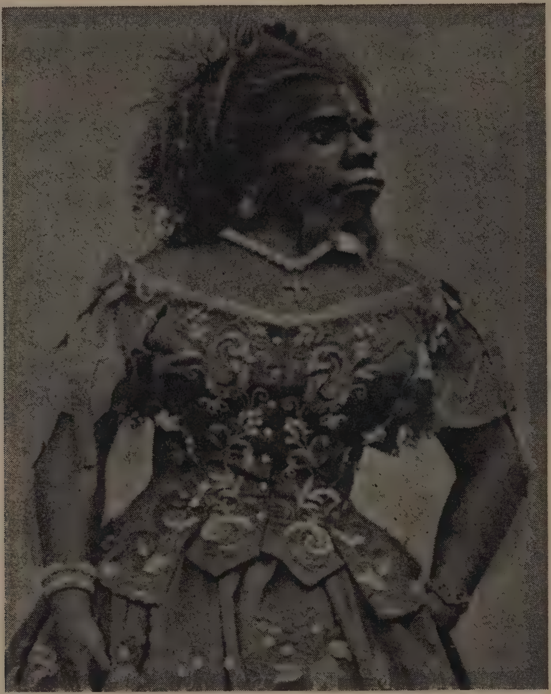

Fig. 566.—Julia Pastrana. From a photograph in the possession of the Anthropological Institute, London.

(B) REACTION OF THE GUMS AND ADJACENT MUCOUS MEMBRANE TO INJURY—INFLAMMATION.

A general inflammation of the mucous tissue of the mouth is called 'stomatitis,' but when the inflammation is limited to the gums it is termed 'gingivitis.'

In this chapter they are dealt with as separate entities; this is done partly to impress on the student that they may be so. But in certain cases, ulcerative gingivitis for example, though confined to the gums when first seen, it may by extension of the process become a stomatitis within relatively few hours.

GINGIVITIS.

This condition may be general, or may be limited to the gum margin—'*marginal gingivitis.*'

Marginal gingivitis is a common affection, and, if not treated, is the forerunner of parodontal disease.

Pathology.—In a mouth that is functional, the constant friction of efficient mastication and the friction of the mucous membrane of the cheeks and lips against the gum margins, assisted no doubt by the free flow of 'currents of saliva,' remove all debris, and keep the gingival margins in a healthy condition. Abnormal conditions, such as mouth-breathing, and functionless teeth, lead to the accumulation of food debris, etc., at the gingival margin; the material thus lodged undergoes fermentative and putrefactive changes, the soft tissue is injured, and inflammatory reaction follows. Such a condition is well marked in 'mouth-breathers.' If the mouth of a child suffering from nasal obstruction be examined, a marginal gingivitis will be found around the anterior maxillary and mandibular teeth—indeed, anterior marginal gingivitis in a child is almost pathognomonic of mouth breathing; while the gingival margin at the posterior part of the mouth will be found quite healthy, provided that the function of mastication is properly performed. In a 'mouth-breather,' the natural friction of the lips against the gums is, to a great extent, in abeyance, with the result that the debris around the teeth is not removed and a condition arises which causes injury to the gingival margin. The gingivitis, so often associated with many fevers, arises, partly at least, from a similar cause. The fevers frequently bring about a condition which induces 'mouth-breathing' and, a 'slop' diet being necessary, the ordinary functions of the mouth are not performed, and food naturally collects at the gingival margins.

The possibility of a dietary deficiency of vitamin C must always be considered in conjunction with or in the absence of other causes.

Although gingivitis is generally the direct result of lodgment of food debris, etc., it may also arise from the formation of toxic material at the gingival margin; notably is this so when mercury is being taken and there is coincident gingival sepsis.

It has been shown by Turner and Drew [1] that in cases of chronic gingivitis bacteria are invariably present. These authors found diphtheroids (fig. 567), streptococci and staphylococci. In one case a spore-bearing bacillus was present. In gum infections they found an entire absence of phagocytosis on the part of the tissues.

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), xii, 7.

Varieties.—The inflammation is usually catarrhal in character. It *may become purulent*, while in certain patients there is a tendency to hyperplasia ; large tags of tissue are formed between the interspaces of the teeth, and the gum encroaches upon the surface of the teeth, which become partly hidden. This form is usually designated '*chronic hyperplasic gingivitis*' (fig. 568). The so-called 'polypus of the gum' is a localised chronic hyperplasic gingivitis.

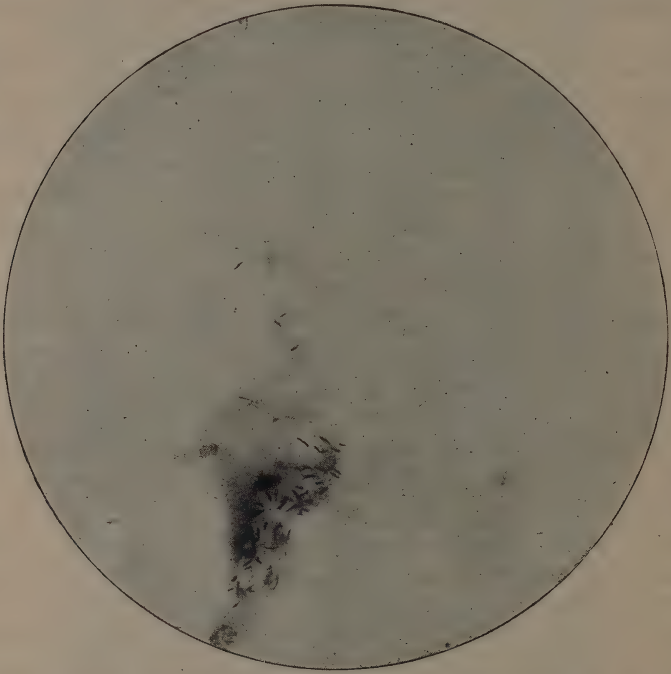

Fig. 567.—Section of gum showing diphtheroid infection.
(Turner and Drew.) (From *Proc. Roy. Soc. Med.* (Odonto. Sec.).

Causes.—(i) *Local.*—Mechanical irritants, such as the accumulation of calculus and food debris at the necks of the teeth ; overhanging edges of fillings and badly adjusted crowns ; the injudicious use of clamps, ligatures, etc., and chemical irritants.

(ii) *General.*—The prolonged administration of metals, such as mercury, arsenic, bismuth or such a drug as belladonna (which lessens the salivary flow). Marginal gingivitis is a frequent accompaniment of certain general diseases, such as diabetes, nephritis, and the possibility of vitamin C deficiency must always be considered.

Signs and Symptoms.—In the acute cases the free margins of the gums are red and painful and bleed freely with the lightest touch. Slight pressure will cause a thick creamy discharge to exude around the necks of the teeth.

In chronic cases—such as chronic hyperplasic gingivitis—dependent on the chronicity of the process the gum is often almost normal in colour, usually there is a crescentic deposit of calculus under the free edge of the gum on the labial aspect which may show through the overlying gum as a dark line; infection is always present in greater or less degree in this abnormal gingival trough.

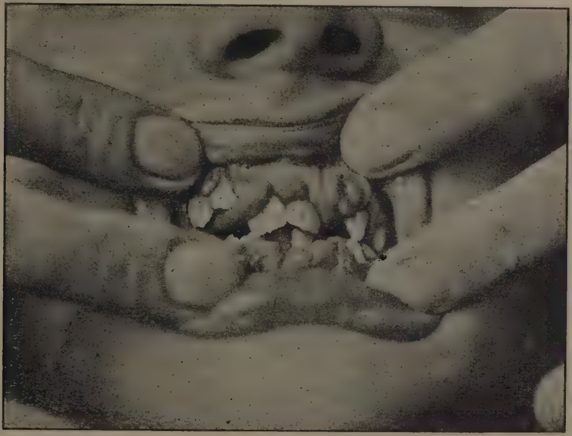

FIG. 568.

Treatment.—The treatment of gingivitis is to remove the cause if possible. When ‘mouth-breathing’ is the cause, it consists first in the restoration of nasal breathing. For this purpose removal of the nasal obstruction alone is not sufficient, as once the habit of mouth-breathing has been acquired it is not easily lost; even when nasal patency is restored it is found that many people, who in childhood have been treated for nasal obstruction, persist in the habit of mouth-breathing when asleep, and this may persist through the greater part of their lives and maintains an otherwise unexplainable and very intractable anterior gingivitis.

It is necessary, therefore, that some form of oral shield be worn at night after nasal patency has been restored in order that this habit may be eradicated (see p. 253). In addition, any deposit of calculus must be removed and the deep gingival troughs irrigated with some mild antiseptic lotion, and astringents such as tannic acid or copper sulphate applied. In due course, when inflammatory œdema has entirely disappeared, it is often found that the fibrosis which always occurs with chronic inflammatory processes has produced a permanent increase or hyperplasia of the gum so that the gingival troughs are still very much deeper than normal and constitute stagnation areas, and in order to reduce them to their normal depth it may be necessary to remove the redundant masses surgically by doing an anterior gingivectomy. This can readily be done under local anæsthesia with such an instrument as a sharp, large spoon excavator or a small knife; the gum is removed along a line festooned to the necks of the teeth and the raw edges epithelialise over in a few days if kept clean (for details see p. 643).

In cases in which vitamin C deficiency may be suspected the vitamin in the form of ascorbic acid may be administered[1] in doses of 300 mg. per day till a urine test (of 5 mg. per 100 ml. of urine) shows that saturation point has been reached.

Acute streptococcal gingivitis is not common; it is characterised by the appearance of a bright red line around the gingival margins. According to A. Bulleid, cultures from gum tissue invariably show a fine growth of streptococcus, usually of the viridans group; gum smears have nothing very characteristic about them, but show many active polymorphs and an increase in fuso-spirochætal and cocco-bacillary flora. Treatment consists of the maintenance of as high a state of oral hygiene as is compatible with the severity of the oral symptoms present, the avoidance of operative interference in the acute stage and the use of drugs of the sulphanilamide group (see p. 456) as indicated.

Lead Poisoning in Relation to the Gum Margins.—Lead may be absorbed into the system via the respiratory tract, skin or alimentary tract; of these the respiratory tract is by far the most important.

After its absorption lead appears in the blood as a dibasic phosphate; most of this is ultimately deposited in the bones as the rather insoluble tribasic phosphate, and over 90 per cent. of the lead stored in the body may be stored in the bones; minute quantities being excreted in the urine and fæces.

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[1] See H. Gordon Campbell and R. P. Cook, *Brit. Med. Journ.*, 1941, i, 360.

In chronic lead poisoning a blue or slate-coloured line often appears on the gums. This was first described by a Frenchman named Grisolle in 1836, and later by an Englishman named Burton in 1840 and is often, though wrongly, called Burton's line. It consists of fine granules in the form of a stippled line within the tissue of the gum about a millimetre from the gingival margin. It is more marked round teeth having infected gingival troughs and occasionally may be found on the mucosa of the

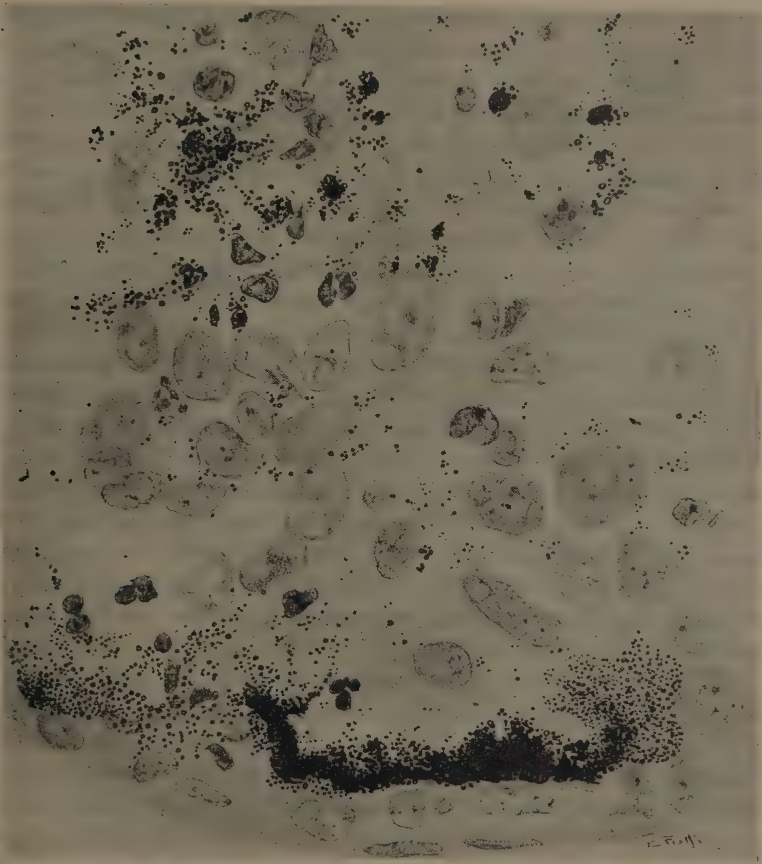

FIG. 569.—Drawing of section showing sub-epithelial position of lead line in gum. (By permission of Professor J. C. Aub and the Williams and Wilkins Co., Baltimore.)

cheek opposite such teeth (fig. 569). The manner in which the line is formed is as follows: It is believed that the soluble lead salts in the circulation are precipitated at the gingival margins by free sulphuretted hydrogen, formed by micro-organisms in septic gingival troughs, as insoluble lead sulphide. The lead sulphide does not *per se* cause a gingivitis; if there are no teeth there is no blue line; a blue line, incidentally, may also be found round the anus. The line is more frequently seen on the mandibular gums than on the maxillary, and in the incisor region than in the molar. In addition to the blue line, lead poisoning usually presents other symptoms, such as colic and wrist-drop, the latter being due to paralysis of the extensor muscles of the wrist. In rare cases the blue line is present, but no other symptoms; in such cases great care must be taken to eliminate all other possible causes. In individuals who take diachylon to produce abortion the characteristic blue line on the gum is frequently present.

It is of interest to note that lead may be deposited in a rather stable form in the calcifying tissues of the body in cases of chronic lead poisoning, and remain there permanently even after removal of the patient from all association with lead. But it is also a fact that pathological conditions such as hyperparathyroidism, or a mild acidosis, and certain drugs may set free lead stored in bones many years after all association with lead has ceased and cause a fresh attack of plumbism; indeed, any condition which increases the blood calcium may similarly set free lead so stored and lead to the same result.

P. R. Howe has recorded that lead may be found in the teeth in case of lead poisoning. In England, S. G. Allen has found it present in the teeth in the ratio of 57 parts per 1,000,000 in a case of chronic lead poisoning extending over ten years in a male aged 45; incidentally, as lead is deposited in calcified tissues only while they are calcifying this proves conclusively that calcification of the dental tissues is still continuing at this period of life. Lead has been found in the bones in cases of chronic lead poisoning in ratios of from 30·1 to 66·66 parts per 1,000,000.

The blue line must be diagnosed from (i) a delicate line of blue at the margins of the gums and teeth, but not involving the gums, which occurs in persons exposed to white lead dust for a few hours. This line is merely lead sulphide deposited upon the gums and disappears on rinsing the mouth.

(ii) A deposit under the margin of the gums, which is found in persons who clean their teeth with charcoal or soot. Under these circumstances small particles may also be forced by the abrasive action of the tooth-brush bristles into the tissues and a permanent tattooing with carbon particles results.

(iii) A deposit similar to (ii), occurring in miners and others exposed to carbon dust.

(iv) The line caused by a semilune of subgingival (serumal) calculus showing darkly through the hyperplasic gum of ‘ mouth-breathing ’ children, and seen chiefly around the maxillary incisors and canine teeth.

(v) The line caused by copper and bismuth poisoning. (See pp. 527 and 537A.)

Treatment consists in removing the patient from the unhealthy surroundings, when the line will generally disappear in three or four weeks, although in severe cases it may persist for a much longer period. Any marginal gingivitis or parodontal disease should receive the appropriate treatment indicated in the pages treating of those diseases.

Chronic gingivitis is usually the result of local irritation. It is frequently seen in patients wearing artificial dentures, and is often due to want of cleanliness. The gums are slightly swollen and are red, congested, and painful. In some cases the palatal rugæ become enlarged and pendulous and by overlapping add to the sepsis. A temporary abandonment of the denture and the use of astringent mouth-washes is sufficient to effect a cure. It may be necessary to excise redundant tissue. The general condition of the patient will need treatment.

Artificial dentures in certain subjects, more especially those of a gouty or rheumatic diathesis, produce a peculiar tenderness of the gums, accompanied by an itching sensation, the patients stating that they can only get relief by removal of the dentures. Local applications are of little use, and the removal of the denture, except, of course, at meals, seems to be the only means of obtaining relief.

A rare form of chronic inflammation of the gums has been described by Arkovy[1] under the name of ‘*gingivitis nudata.*’ The disease usually has its seat in the roof of the mouth and spreads until it involves the interdental papillæ, or it may be limited to the latter. It appears at all stages of life and may continue for one or two years. With the naked eye it is difficult to locate the junction of the affected with the non-affected part, as the edges are not pronounced and the colour of the gums is unaltered. Histologically, there is an entire absence of the epithelial covering of the sub-mucous tissue, leaving the papillæ exposed.

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[1] *Dental Cosmos*, 1893, October.

The etiology is obscure: occasionally it may be traced to scalding. Dentures with rough surfaces, or which do not fit accurately, may contribute to the production of the disease. The symptoms are a feeling of 'continual burning with sensitiveness of touch, especially during meal-times.'

Treatment should aim at restoring a layer of epithelium over the denuded sub-mucous tissue. All irritants, whether in the form of foods or otherwise, should be avoided. The foods taken must be of a mucilaginous and slippery character, and all medicaments must be administered in a similar condition.

Ulcerative Gingivitis.—See p. 517.

'Polypus' of the Gum.—'Polypus' of the gum is the name given to a local inflammatory hyperplasia of that tissue caused by irritation.

Etiology.—This condition is generally found in connection with the sharp cervical edge of a carious cavity or root, or may be brought about by the presence of calculus.

Appearances.—In character and microscopical appearance a polypus resembles inflamed gum tissue. The growth really starts as a simple inflammatory swelling of the gum; this increases and becomes hyperplasic and pedunculated, and, if the cause is not removed, becomes so enlarged that it encroaches upon the cavity and eventually completely fills it. Should the hyperplasic tissue come in contact with the opposing teeth, ulceration will ensue and give rise to considerable pain.

It may here be pointed out that the pathology of a 'polypus' of the gum is identical with that of a fibrous epulis (see p. 948); each, at its commencement, is an inflammatory hyperplasia, but the 'polypus' usually occupies an interstitial cavity.

The fibres of the pedicle of a 'polypus' are often found to have their origin from the periodontal membrane, so that when a tooth, the interstitial cavity of which is occupied by a 'polypus,' is removed, the 'polypus' comes away with the tooth with its pedicle fibres still in continuity with their periodontal origin.

The same is true of the fibrous epulis; its pedicle often has origin from the periodontal membranes, so that on extraction of the tooth it comes away with its attachment unbroken (see fig. 940).

Diagnosis.—When the polypus occupies the cavity, care must be taken to diagnose it from a polypus of the pulp. The diagnosis is not difficult, as in one case the pedicle of the growth will be in connection with the pulp chamber, while in the other it can be traced to the gum or periodontal membrane between the teeth. The polypus of the pulp is not painful to the touch, whereas polypus of the gum is extremely painful to pressure.

The treatment consists in removing the source of irritation, but, in addition, it is generally advisable to snip off the growth, or, if the growth is in connection with a cavity, it is better to remove the growth with the electric cautery or with a sharp spoon excavator after the injection nearby of a few minims of 2 per cent. novocaine solution. The filling of the cavity will remove the source of irritation so it will not recur.

STOMATITIS.

Catarrhal Stomatitis.—This form is generally associated with inflammation of the throat or nose, and probably arises from the same cause as catarrh of those parts. It is frequently seen in the course of the acute infective fevers and gastro-intestinal disturbances. The excessive use of tobacco, or too hot or too highly seasoned foods, may be cited as causes.

Signs and Symptoms.—The serum from the congested mucous membrane filters into the sub-epithelial space. There is an increased production of surface epithelium and also of mucus. The mucus continues to accumulate and, with the serum, forms the peculiar sticky discharge seen in these cases. In more severe cases the epithelium may be detached in large masses, the sub-mucous tissue becomes greatly infiltrated with leucocytes and the whole mucosa swollen, giving rise to a purulent or muco-purulent condition. The margins of the gums become acutely inflamed and painful to the slightest touch. In the early stages the gums are dry, but this is soon followed by an excessive secretion. The portion of gum attached to the margin of the alveolar process is pale, while the reflection of the gum from this point on to the cheek will show the vessels to be congested. The gums appear whitish and mottled, and pus is generally seen welling up from the sulci around the teeth.

The mottled appearance of the gums is due to the fact that in inflammation the epithelium proliferates and appears whitish and sodden, but through the friction which occurs between the gums and the cheeks the epithelium covering the papillæ of the gums is rubbed off and leaves reddish patches here and there. The breath is fœtid, the patient has a sensation of heat and pain, and the taste is impaired. The tongue is furred, there is a loss of appetite, derangement of the bowels, and a feeling of malaise.

Treatment.—The cause should, if possible, be removed, antiseptic and astringent mouth-washes should be used and the general condition of the patient treated by appropriate remedies.

Follicular Stomatitis.—This inflammation is similar to an ordinary herpetic eruption. A cluster of vesicles first appears ; the vesicles break down, coalesce and form a small circular and well-defined ulcer. This ulcer is surrounded by a zone of redness and is extremely painful. The ulcers are said to occur more frequently near the frænum of the lip, on the under surface of the tongue, and in the sulcus between the gums and the lip. The small round punched-out ulcers met with in the cheeks are probably a variety of this form. Follicular stomatitis occurs in adults less frequently than in children. It is often associated with gastro-intestinal disturbance.

The *treatment* consists in the application of an astringent solution, and, in intractable cases, the ulcers may be touched with a crystal of sulphate of copper or nitrate of silver. The general condition of the patient must also be treated.

Aphthous Stomatitis.—This form rarely occurs in adults. It is characterised by the formation of fibrinous deposits on and under the epithelium, and is considered by some authors to be contagious. It is frequently met with in rachitic and weakly children and is most common during the periods of dentition. In adults it occurs in those debilitated by illness, or may be associated with general inflammatory conditions. In women it occurs during menstruation, pregnancy, and during the puerperal period.

Bacteriology.—Of ten cases of aphthous stomatitis examined by K. W. Goadby, there was present in six of them a streptococcus which had the characters of the *Streptococcus fœcalis*, and not of the ordinary *Streptococcus brevis* of the mouth.

Signs and Symptoms.—Small yellowish-white patches, slightly elevated and extremely sensitive, are present on the mucous membrane. The patches are surrounded by a zone of inflammation. They have a tendency to spread and coalesce, forming large patches. When not associated with any active general disease, constitutional symptoms may be present, namely, slight elevation of temperature, thirst, and loss of appetite.

Treatment.—Locally, a mouth-wash of chlorate of potash should be used, and the mouth thoroughly cleansed after each meal. If the aphthæ persist, they may be treated with nitrate of silver. The constitutional condition also requires attention.

In cattle a form of aphthous stomatitis appears in an epidemic form and is commonly known as 'foot-and-mouth disease.'—It is communicable to man. In man it is an eruptive fever running a fixed and definite course, usually of eight or ten days, and is characterised by a vesicular eruption in the mouth, on the lips, gums, and tongue, as well as on the hands and feet. In the early stages of the disease there is a headache and loss of appetite, with a quickly rising temperature. Pustules appear in the mucous membranes of the mouth, more frequently on the lips and the tongue than on the hard palate and the throat. After a few days the pustules burst, leaving eroded spaces with greyish-yellow surface, which during the second week commence to heal. In the acute stage, the patient suffers from a sensation of heat and burning in the mouth, and the cervical and salivary glands swell, producing severe dysphagia.

Details of two cases noted by Hobart Nixon [1] are as follows :—

> In April, 1937, an auctioneer aged 24 consulted me casually for a 'sore mouth.' There was slight gingivitis, and there were three small bullæ the size of split peas in the buccal cavity ; the temperature was normal. The following day the bullæ, which had become larger, were punctured by the teeth, and two fresh ones appeared ; the temperature was 99·2° F. Next day there were numerous bullæ and painful ulcers, and the temperature was 100·2° F. ; but constitutional symptoms were surprisingly slight considering the filthy appearance of the mouth. The case was transferred to hospital, where the diagnosis of foot-and-mouth disease was confirmed. The Ministry of Agriculture were assisted in their investigations by Professor J. A. Nixon of Bristol, but up to date no trace of the infected cattle has been found.
>
> Comparison with a case reported ten years ago is interesting. The daughter of a local farmer had only slight signs in the mouth, but there were extensive lesions of the hands ; examination of the cattle in the yard brought to light early 'limping' and 'slobbering,' and the diagnosis in three of the beasts was soon confirmed by the Ministry officials, and an extensive epidemic efficiently controlled.

Ulcerative Gingivitis—Ulcerative Stomatitis—Vincent's Angina.—This condition is most frequently met with in children. It may prevail in an epidemic form in institutions, and to a certain extent has a seasonal incidence, being most prevalent in the first cold weather in autumn. Want of cleanliness, faulty hygienic surroundings, and general ill-health favour the development of the disease. It occurs also in adults ; a notable outbreak occurred among troops on the Western Front during the Great War, the outbreak reaching its peak in the autumn of 1918, when it was known by the name of ‘ trench mouth.’

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[1] *Brit. Med. Journ.*, 1937, 2, 139.

In the early stages the gums are swollen and congested. The inflammation and ulceration commence at the free margin of the gums, these when ulcerated bleed readily and present a ragged margin of necrotic tissue (fig. 570), the infection spreads rapidly and may within twenty-four to forty-eight hours involve both jaws, and possibly extend to the hard palate and the floor of the mouth ; the adjacent mucous surface of the cheek may become attacked, possibly from contact, and in untreated cases the extension of the process may lead to extensive sloughing, necrosis of bone and loosening and loss of teeth, and even the death of the patient.

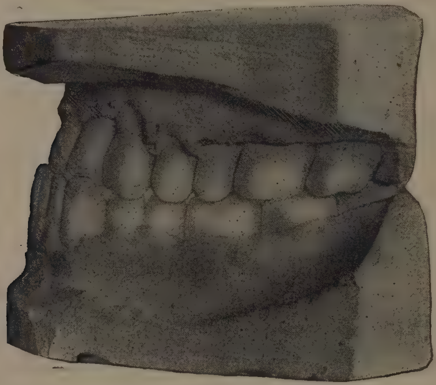

Fig. 570. (From *Dental Cosmos.*)

The surfaces of the ulcers are covered with yellowish or greenish-yellow sloughs which readily become detached, exposing a freely bleeding surface ; the ulcerated surface may remain superficial or extend more deeply and lead to exposure and ultimate necrosis of bone. The breath is very offensive and there is a rise of temperature from 100° to 103° ; the lymphatic glands draining the area involved are enlarged.

In children one occasionally finds one side of the mouth only is involved, and this may remain so possibly for a week or more, and then, as the condition clears up, it appears on the other side ;

children, in spite of the ulcerated condition, offensive breath, and rise of temperature, will not infrequently say that they feel quite well. When the disease occurs about the age that the deciduous teeth are normally shed these frequently become loosened and partly detached, so that they are an additional cause of irritation and the retention of discharge. There is strong evidence that the disease is communicable.

The name of Vincent's angina is applied rather to those cases presenting faucial lesions; these vary in type, some cases being relatively mild or diphtheroid, superficial and limited in extent, others being severe and of ulcero-membranous type in which the process extends more rapidly and deeply.

Taylor and McKinstry[1] are of opinion that the primary lesion is in the gums, and that the throat, pharynx, etc., are infected from this source.

Any condition which lowers the vitality of the individual may predispose to this disease; such conditions may be environmental, such as prolonged and intense worry and anxiety or life under unsanitary and exposed conditions, or they may be of the nature of absence from the diet of antiscorbutic vitamins; support is given to this latter view by D. Clewer,[2] who tabulated fifty cases as follows:—

Cases receiving no fresh vegetable ration	41
,, ,, vegetable ration once a week	2
,, ,, ,, ,, twice ,, ...	3
,, ,, ,, ,, thrice ,, ...	3
,, ,, ,, ,, more than thrice a week ...	1

Bacteriology.—In this disease, the *Bacillus fusiformis* and *Spirochœta dentium* are always present in great numbers, living in symbiosis; and observers who have examined such cases have generally come to the conclusion that the fusiform bacillus and the spirochæte are the causative organisms.

In the mild type the bacillus predominates, in the severe type the spirochæte is 'much more frequent and may even be present in greater numbers than the bacillus.'

R. Tunnicliff[3] in an examination of freshly excised tonsils affected with Vincent's angina found enormous numbers of *B. fusiformis* and spirochætes in the zone separating the external necrotic epithelium and the living tissue. The organisms were invading the normal tissue, the spirochætes being more abundant and in advance of the bacilli. 'In the line of advancing necrosis there appeared to be no association with cocci and bacilli, which were abundantly present in the exudate on the surface of the tonsils.'

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[1] *Brit. Med. Journ.*, 1917, i, 421.

[2] *Brit. Dent. Journ.*, 1919, xl, 749.

[3] *Journ. Infect. Diseases*, xxv, 132.

Guezette[1] considers that ulcerative stomatitis is closely related to noma. The most constant feature in three cases he examined was the presence of a spirillum and a bacillus. The discharge from a case he injected into his own lip and found that the spirillum speedily disappeared whilst the associated streptococcus flourished freely.

Treatment.[2]—Treatment should be local and general and along the following lines. As the disease is communicable, cases occurring in residential schools and such-like collections of individuals should be quarantined. While the temperature is raised the patient should be kept in bed, and food and drinking utensils should be sterilised after use. The bowels should be kept open. The surface of the ulcers should be cleaned with 2 volume peroxide of hydrogen and strong tincture of iodine applied locally; this should be followed by the application of chromic acid, in strength from 10 to 20 gr. to the ounce of water, used warm and dabbed on to the ulcerated surfaces by means of cotton wool swabs, and this may occasionally be alternated with warm carbolic acid lotion of 1 in 100 to 1 in 200 strength in saline, to act not only as an antiseptic but as an analgesic.

Chlorate of potash may be used as a mouth-wash, gr. 10 to 1 oz. of water, or may be given internally in 15 to 20 gr. doses for adults, and proportionately less for children[3]; its action[4] is believed to be due to nascent oxygen acting as a stimulant and antiseptic to the septic tissues by which it is easily decomposed; moreover, as some of it is excreted in the saliva its action is kept local and therefore more constant.

It is wiser *not* to remove loose or septic teeth at the acute stage of the disease, as one is thereby laying open fresh paths for infection ; but if, in cases of partly absorbed deciduous teeth, they are so loose as to be partly detached and possibly exposing the successive teeth, it may be wiser to remove them, as they act as irritants and retain discharge.

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[1] *Archiv. de la Sci. Med.*, xxiii, No. 1.

[2] More recently the use of penicillin lozenges of 500 units, without any other preliminary treatment, has been found to give excellent results, not only in this, but in many other acute oral infections; the lozenges are allowed to dissolve slowly in the mouth, each one lasting up to two hours; pain is frequently absent after 24 hours use; after several days, scaling and other hygienic measures may also be undertaken. Similar results from the use of penicillin used intramuscularly have also been obtained.

[3] Dosage for children under 12 years of age is usually $\frac{\text{age}}{\text{age} + 12}$ that of the dose for the adult (except in the case of opium).

[4] This action of chlorate of potash is entirely denied by some pharmacologists. (E, S.)

As arsenic is believed to be a specific for spirochætes, doses of 2–3 minims of liquor arsenicalis hydrochlor may also be given at this stage, or one of the organic preparations of arsenic such as stovarsol.

If the mouth remains very offensive potassium permanganate, the official liquor diluted 1 in 50, may be used as a mouth-wash, being deodorant as well as antiseptic. Under the above treatment the acute stage is usually soon over, though it will often persist for a week or even longer, and be followed by a persistent chronic condition which requires careful hygienic treatment for the subsequent two or three weeks.

As soon as the temperature is normal the patient should be up and out in the sunlight as much as possible, and a nourishing diet given, together with a tonic such as :—

℞	Quininæ hydrochloridi	gr. i
	Liquor strychninæ hydrochloridi	♏. iij
	Liquor arsenicalis hydrochloridi	♏. iij
	Magnesii sulphatis	gr. xxx
	Acidi hydrochloridi dil.	♏. i
	Aquam ad	℥ss

(One tablespoonful to be taken three times a day after meals, adult dose.)

In cases in which vitamin C deficiency may be suspected the vitamin in the form of ascorbic acid may be administered in doses of 300 mg. per day till a urine test (of 5 mg. per 100 ml. of urine) shows that saturation point has been reached.

Following the view of the specificity of arsenic for spirochætes, the intravenous administration of organic arsenic compounds has been successfully used by some practitioners. It is, of course, necessary to determine by microscopic film examination that spirochætes are among the organisms present before such a course of treatment is decided on. According to J. Carter, who has used this method extensively, remarkable results have been achieved by the intravenous administration of the trivalent arsenical compound Neoarsphenamine (N.A.B.) (synonyms: Neosalvarsan, Novarsenobillon, and Neokharsivan)—a condensation product of formaldehyde sulphoxylate of sodium and salvarsan.

Extreme caution must be exercised in its administration, bearing in mind the contra-indications to its use, *i.e.* extensive visceral disease (kidneys, spleen, liver, and lungs), cardio-vascular or cerebro-spinal affections, dermatoses, and known cases of hypersensitivity to arsphenamines.

The technique of injection must be rigorously adhered to, for N.A.B. undergoes oxidation fairly readily and forms products of high toxicity; therefore, in charging a syringe for injection only cold water should be used previously distilled and sterilised; undue mechanical shaking which introduces air bubbles, and thus favours oxidation, must also be avoided.

The injection is usually introduced into the median basilic vein, and must be conducted slowly, otherwise much pain and shock will attend its administration, characterised by unpleasant effects such as headache, malaise, vomiting, rigors, or fainting attacks. The most important of the immediate toxic effects are the vaso-motor reactions which appear either during or within a few hours of the injection, *i.e.* chill rigors, followed by pyrexia and headache, together with a fall in blood pressure; this may be followed by urticaria. These reactions, although alarming, are seldom serious, for in the treatment of Vincent's angina only one injection is given, seldom two, and then only in very stubborn and extremely bad cases.

The amount used for an adult is 0·45 grm., and only 0·15–0·3 grm. for younger people. This gives excellent results, the majority without constitutional upsets.

After about forty-eight hours (though this is somewhat variable) considerable hyperæmia of the oral tissues is noticed which persists for several days, the sloughs are then slowly separated accompanied with much salivation, and in the course of seven to ten days complete resolution takes place, the oral tissues returning to normal.

It is most important that mouth-breathing, if present, should be corrected; as long as it is present the condition is extremely difficult to control.

During the Great War, and particularly towards the end, this disease was prevalent in epidemic form among troops. The local application of ipecacuanha and arsenic was much used in its treatment; this may be made up as follows:—

℞	Tinct. ipecacuanhæ	℥ss
	Glycerini	ʒj
	Liq. arsenicalis	ad ℥j

In intractable cases the local application of salvarsan has been recommended, the drug being applied to the pocket on a swab moistened with glycerine.

Some cases respond to treatment very much more readily than do others ; one sometimes finds this disease rather intractable to treatment.

Gangrenous Stomatitis : Noma ; Cancrum Oris.—This very serious disease is a rapidly spreading gangrenous inflammation which usually attacks the cheeks, though existing infection in the mouth may decide the incidence and site of the oral lesion, and occurs in children from two to six years old. The disease may, however, occur in adults.[1] It is more common in girls than in boys. It is frequently seen in those just recovering from one of the acute specific fevers. Unhygienic surroundings and weakening of the resistance are predisposing causes. The disease may start either in the substance of the cheek, or in the mucous membrane, the latter being the more usual situation. The cheek becomes hard, brawny, and very swollen, a dark red colour showing in the centre, the surrounding parts being œdematous. At this stage, if the mouth be examined, an ulcer will be seen on the mucous surface of the cheek corresponding to the dark spot on the cheek. The ulceration, or rather sloughing, leads to perforation of the cheek, and, if the disease still pursues its course, the soft parts rapidly become gangrenous and there may be necrosis of bone. The child becomes exhausted, delirious, and eventually dies of exhaustion, septicæmia, or some septic affection of the lungs.

Most authors think that the difference between cancrum oris and ulcerative stomatitis is only one of degree. The disease has been said to be caused by thrombosis of the capillaries, induced by the presence of a specific micro-organism, and to be similar to the gangrenous inflammation known as noma, which occurs upon the female genitals.

The disease in rare instances may commence at the gingival margin. A case in point is recorded by C. Lockyer.[2] The patient, a youth aged eighteen, removed three roots of a tooth by means of a wooden penholder sharpened and used as an elevator. Four days subsequently the face began to swell, gangrene ensued and spread rapidly over the palate and the alveolar process, the patient eventually succumbing to general septic infection (fig. 571).

The *prognosis* of cancrum oris is bad. The *treatment* consists in carefully drying the soft parts, removing all gangrenous portions, and cauterising the remaining surface with nitric acid, or the actual cautery. Grey Turner[1] advocates excising the involved area with the diathermy knife, cutting through healthy tissue about a quarter of an inch beyond the spreading edge. Areas which cannot be excised in this way should be treated with the coagulating current. The general treatment should consist in supporting the patient's strength with a plentiful supply of nutritious remedies. It has been suggested that free excision of the gangrenous surfaces would be an effective treatment, and in cases recorded it has proved beneficial. Corrosive sublimate locally applied has been used; it has proved successful in three cases recorded by Kingsford.[2] Disinfectant mouth-washes must be prescribed, and the raw surfaces carefully dressed with antiseptics. The disease may spread until the

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[1] *Lancet*, 1921, December 21 ,1730.

[2] *Brit. Dent. Journ.*, 1898, xix, 607.

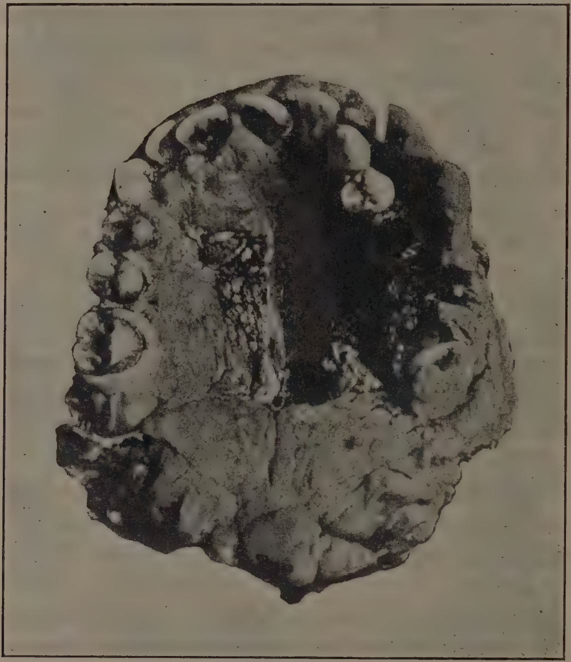

FIG. 571.—A case of gangrenous stomatitis. (Museum of Charing Cross Hospital.)

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[1] *Brit. Med. Journ.*, 1937, 2, 150.

[2] *Lancet*, 1889, May 4.

whole side of the face disappears, the cavity extending from the nose to the ear, and from the lips to the upper eyelid.

Parasitic Stomatitis.

Thrush (Monilil).—Thrush is a parasitic inflammation of the mucous membrane of the mouth. It is common in infants, but may occur in adults, when it is known as monilil, and, in the latter, is generally associated either with one of the acute specific fevers, or with chronic wasting diseases, such as phthisis. When associated with phthisis it generally proves fatal. The fungus develops in the upper layers of the mucosa, the filaments forming a dense network among the epithelial cells. In this manner the mucous membrane becomes covered with numerous white spots, which are firmly adherent, and, when removed, a deep red colour is revealed. These spots appear mostly near the angles of the mouth and on the tongue, but may occur elsewhere in the oral cavity, the affection spreading at times to the pharynx and œsophagus. The spots are about the size of a pin's head, are circular in form, and they gradually coalesce and form larger patches, giving rise to the appearance of a false membrane with a slightly yellowish aspect. These patches come away of their own accord, leaving a reddish surface beneath. The patches are found to consist of epithelium and fat, together with sporules of the vegetable parasite which causes the disease.

It is usually acknowledged that thrush is due to the *Saccharomyces albicans*. Castellani,[1] however, states that in the tropics, at any rate, thrush is not caused by one fungus only, but by a number of different fungi, and that in reality the term thrush does not cover one affection, but a group of clinically similar conditions due to various organisms. He describes eleven types of Monilia found in thrush. Fungi of the genus Monilia are most common, and to this genus belong *Oïdium albicans* or *Monilia albicans*. Fungi of the genera Hemispora, Willia, Endomyces, and Saccharomyces are also met with.

When children are affected by thrush they are generally found to be out of health, the bowels relaxed, the evacuations green and sour. The motions are generally acrid, and irritate the margins of the anus, giving rise to an erythematous blush over the buttock, the appearance of the edges of the anus being similar to that seen in the mouth. Sucking and deglutition are impaired by the condition of the mouth, and the child will usually be in a state of drowsiness and torpor. Many cases of thrush in infants are traceable to the use of dirty feeding-bottles.

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[1] *Lancet*, 1920, April 24, 895.

The *treatment* of thrush may be divided into local and general. The *local* treatment consists in carefully wiping the mouth with soft lint after each meal, care being taken to burn the lint after use. The exposed surfaces thus left are touched with a solution of 3 dr. of borax to 1 oz. of water. In severer cases, it will be advisable to use nitrate of silver, 5 gr. to the ounce, or dilute carbolic acid in glycerine. It is needless to say that attention should be given to the condition of the feeding-bottle. The *general* treatment should consist in careful attention to the diet, with the administration of a mild aperient.

Due to the Aspergillus nigrescens.—An inflammatory condition due to the presence of the *Aspergillus nigrescens* has been reported.[1]

The following is a brief account of the case: 'A small ulcer first appeared on the middle line of the roof of the mouth, about half-way between the incisors and the soft palate. The patch increased slowly in size, and others formed in the neighbourhood, the condition appearing like a lumpy patch extending from just behind the incisors to within one-fourth of an inch of the soft palate. Cup-shaped elevations on the soft palate appeared on either side of the middle line. A firmly attached membrane, giving rise to hæmorrhage when forcibly removed, covered the areas. The colour of the recent deposit suggested the sulphur-coloured scutula of favus; where it had remained undisturbed it was darker. With low power the growth was recognised under the microscope as a fungus differing from the achorion. The mycelium network was composed of delicate fibres, bearing perpendicular fructifying hyphæ. Scattered over the field were a number of fruit receptacles and a few spores. The manner of fructifying showed that the fungus did not belong to the oïdium, but to the ascomycetous genus. Cultures showed the *Aspergillus nigrescens*. Upon applying 25 per cent. ethereal solution of pyrozone, improvement was immediately noticed. The pseudo-membrane disappeared and new patches ceased forming. After seven weeks' treatment the patient was well.

'The spores were supposed to have been implanted in the mouth through the medium of cheese, strong and mouldy varieties, of which the patient was very fond of eating.'

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[1] *Medical Record*, October 1896 (*Dental Digest*, 1896, 641).

Mercurial Stomatitis, and Stomatitis of other Metallic Origins.—Prolonged use or overdosage with mercury frequently used to lead to gingivitis which spread rapidly to contiguous parts. The more careful use of mercury, chiefly in the course of anti-syphilitic treatment, coupled with the fact that efficient dental treatment is often insisted on before such a course is commenced, has made the incidence of these cases almost a rarity, and if seen nowadays, it is usually only in the incipient stages. Occasionally they may be seen in patients having an idiosyncrasy to mercury salts.

The symptoms caused by mercurialism (*i.e.* chronic poisoning by mercury) are first, fœtor of the breath and soreness of the gums, soon followed by a disagreeable metallic taste in the mouth and the gums becoming swollen and soft and bleeding readily; this is followed by intense salivation, the saliva being thick and viscid. The symptoms gradually become more marked and the tongue swells. The teeth then become loose, salivation increases, and the salivary glands become enlarged and tender and there is a slight rise in temperature. Unless the progress of the disease is checked the teeth then fall out and there follow extensive ulceration of the mouth and tongue, necrosis of the jaws, great weakness, emaciation and anæmia, liability to hæmorrhages, exhaustion, and death. (See also p. 934.)

The clinical appearances are, in the earlier stages, deeply congested gums, particularly near the free edges, and in parts presenting a whitish or whitish-mottled appearance; ulceration and sloughing commence near the margin of the gum. The inflammation spreads rapidly to the cheeks, tongue, floor of the mouth, and salivary glands, and there is much pain on swallowing, speaking, and on moving the jaws.

The *signs and symptoms* in **bismuth** stomatitis are very similar, but in this case a purplish line following more or less the contours of the teeth appears on the gums and similarly coloured spots on the floor of the mouth and cheeks; necrosis of tissue commences at those spots. The breath has an intense smell of onions or even like acetylene. It is probable that the purplish line on the gums and spots in the mouth are due to the deposit of the sulphide of bismuth, and occur there in a similar manner to the bluish line which appears in cases of chronic lead-poisoning.

With regard to the pathogenesis of mercurial stomatitis, Alonkvist[1] considered that the metal circulating in the blood was deposited in the vessels of the gum margin in the form of the sulphide by the action of hydrogen sulphide formed from proteid matter around the gum margins; the mercuric sulphide acting as an irritant and so rendering the tissues susceptible to infection. J. E. McDonagh maintains there is nothing specific in mercurial stomatitis. He states[2] that 'it is merely a sign of metallic intoxication and can follow equally well the over-use of other metals, such as aluminium, antimony, arsenic, bismuth, copper, manganese, thorium, etc. A metal produces intoxication when it undergoes oxidation, and this is only when it has caused such a dispersion of the protein particles in the serum as to render the same acid in character. Stomatitis is one of the symptoms of this increased acidness. Non-metals, belonging to the sulphur group of elements, which includes sulphur, selenium, and tellurium, cause condensation of the protein particles, which has the effect of increasing their baseness. Furthermore, these non-metals prevent metals from becoming oxidised; instead, they manufacture the medium upon which metals work to increase the oxidising action of the protein particles. It thus comes about that metals are antidotes in non-metallic poisoning and non-metals in metallic poisoning. Hence the introduction of intramine (di-ortho-di-amino-thio-benzene) for use in mercurial and arsenical intoxication. Owing to the fact that intramine cannot be readily injected intravenously and that it causes pain when injected intravenously, I have replaced it by contramine (di-ethyl-ammonium di-ethyl-di-thio-carbamate). Contramine is readily soluble in water, easy to inject, painless, and very much more efficacious than intramine. Contramine should be used in 0·25–0·5 grm. doses.'

D. Clewer[3] refers to eighty-eight cases of mercurial stomatitis treated with intramine. Marked relief was obtained in twenty-four hours in forty-three cases, and in the course of a few days in forty-four cases.

Gonococcal Stomatitis.—This is a rare condition; infection occurs in the newly born during transit of the maternal passages, but in adults usually as the result of direct infection, though it is thought that it may occasionally be metastatic in origin. The incubation period in adults is believed to vary from one to seven days. In a case recorded by Frazer and Menton[1] in which the organism was recovered from the superficial exudate in the mouth : 'The buccal mucosa, anterior half of the tongue, the gums and the tonsils were coated with a greyish adherent membrane. In places the membrane had sloughed off, leaving bright red areas with numerous bleeding points. These superficial ulcers were more numerous in the neighbourhood of the molar teeth than elsewhere. The gums were swollen and retracted. Pain in the mouth was a prominent symptom. The temperature was 101° F. While under treatment the membrane sloughed off in parts showing red mucosa beneath without ulceration. The redness gradually faded till the normal colour was resumed.'

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[1] See 'Mercurial Stomatitis: Present Status of the Question,' Raymond Lulle, *Le Progrès Dentaire*, 1907, September.

[2] Private communication.

[3] *Brit. Dent. Journ.*, 1921, xlii, 769.

Oral treatment consisted of the frequent use of a mouth-wash of 1 in 5000 potassium permanganate ; the gonococcus, being easily accessible in the mouth, is apparently easily killed by a weak solution of almost any disinfectant.

Koplik's Spots.—These are small bluish-white spots due to necrosed epithelium about the size of a pin's head surrounded by a reddish areola, found on the mucous membrane of the cheek, and best seen opposite the premolar teeth during the prodromal stage of measles. The areolæ may become confluent, and the spots spread over the mucous membrane of the cheeks and inside the lips. The spots usually fade with the appearance of the rash.

(C) LEUCOPLAKIA.

(See also Chapter XXXV.)

Leucoplakia of the cheeks and the palate is of frequent occurrence in syphilitics and those addicted to heavy smoking. It is more usually seen in the muco-periosteum covering the back of the hard palate than on the gums, and in marked cases presents the appearance of 'ichthyosis.' At times the condition may be limited, and give rise to a definite swelling of a white, cauliflower-like growth. H. P. Pickerill[2] has recorded a case of this character. On the tissue being removed and examined microscopically, the growth was found to consist of epithelial cells, which, instead of becoming squamous and then desquamating, had become swollen and hardened and remained *in situ* (fig. 572). The sub-mucous tissue was normal. This author considers the

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[1] *Brit. Med. Journ.*, 1931, i, 1020.

[2] *Brit. Dent. Journ.*, 1909, xxx, 733.

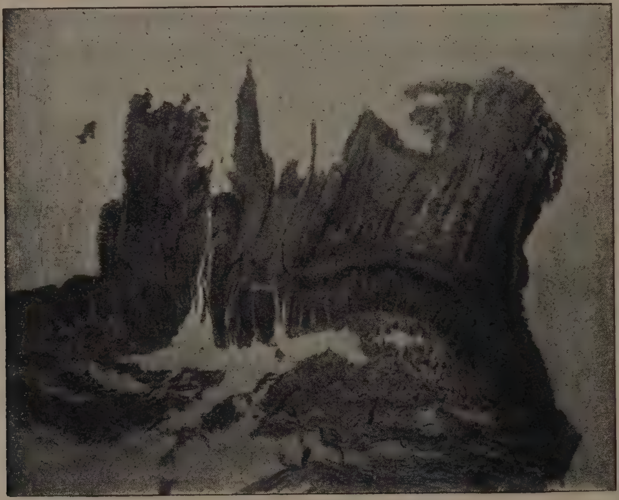

FIG. 572. (From *Brit. Dent. Journ.*)

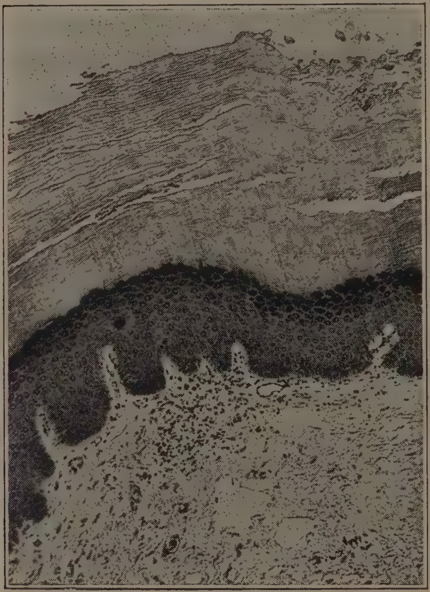

FIG. 573.—Severe leukoplakia of buccal mucosa. (Low power; hemalum-erythrosin-saffron stain.) (From *Journ. Amer. Dent. Assoc.*)

condition similar to the 'pre-cancerous' conditions of the tongue described by H. T. Butlin.[1]

When the lesions occur on the mucous surface of the cheek there is thickening of the stratum corneum, and a sub-epithelial infiltration with small round cells which is not, however, specific (fig. 573). (See also p. 998.)

(D) PEMPHIGUS.

The eruption of pemphigus may occur in the mouth, and one rare form, *Pemphigus vegetans*, may remain localised to the mouth and adjacent cavities. The following case[2] will illustrate this condition.

The patient was a man, aged seventy-two, who complained of soreness in his mouth and inability to take solid food. On the roof of the mouth and on the epiglottis were patches of false membrane of considerable thickness, which, when removed, left a raw, bleeding surface. Some carious teeth were extracted and antiseptics used, but blebs formed on the roof of the mouth, the soft palate, the cheeks, under the tongue, and on the posterior wall of the pharynx. Bacteriological examination of the membranes gave negative results. There was neither fœtor nor salivation. Whenever the patient attempted to masticate solid food a fresh crop of blebs appeared.

(E) PURPURA.

Purpura, *per se*, is a symptom rather than a disease, and many acute infections, particularly those of a more severe type, may give rise to local hæmorrhages, either subcutaneous or under the mucous membrane lining the mouth. The various forms of purpura, whether of toxic or microbic origin, have been described as external manifestations of internal disorders.

In the severe form known as 'purpura hæmorrhagica' the gums and mucous membranes may be the seat of hæmorrhages. A similar condition is occasionally seen in patients who are the subjects of hæmophilia, spontaneous hæmorrhage occurring at the margin of the gums.

The treatment of purpura belongs to the domain of general medicine. The hæmorrhage from the gums can be treated by the use of astringents such as tannic acid, the application being combined with a certain amount of pressure (see also p. 875 *et seq.* for treatment of hæmorrhage).

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[1] *Brit. Med. Journ.*, 1909, January 9.

[2] Lewis Maller, *New York Med. Journ.*, 1898, July 3.

At the same time the dental surgeon is concerned in the maintenance of oral and dental hygiene, which is, in these and allied cases, an all-important factor ; the proof of this lies in the fact that in the absence of teeth oral symptoms are frequently absent. Owing to the acute and often severe nature of the infections which give rise to purpuric hæmorrhages it is wise to avoid any radical treatment as far as possible during the acute stages ; the risk of further hæmorrhage and of laying open fresh paths for infection in tissues, the vitality of which is already so much lowered, strongly contra-indicates such treatment.

(F) ACUTE LYMPHATIC LEUKÆMIA AND ACUTE MYELOID LEUKÆMIA.

These diseases have been likened to neoplasms of the blood, and are very similar to each other in that the prevailing abnormal cells of the two types are probably derived from the same primitive cell.

They are often first brought to notice by reason of the swelling of the gum and occasional gingivitis and sepsis (which may be intense) around the necks of the teeth, so that sufferers may seek the dental surgeon's aid before that of the physician. In some cases there is persistent hæmorrhagic oozing from the gums. The gum may present a local swelling, usually on the buccal aspect, sometimes accompanied by the loosening of one or two teeth in its immediate neighbourhood. The swelling may at first be of normal colour, but often presents a mottled appearance two or three days later and may even become almost black, and the discoloration may extend on to the adjacent cheek and along the gum margin.

Usually the patient appears anæmic, but this sign may be absent. It is common for complaint of tiredness of several days' duration to be made, the glands are commonly not obviously enlarged and are soft and sometimes not palpable ; an important sign is that there may be petechiæ or a petechial rash on the body.

These cases are of great importance to the dental surgeon, for if teeth are extracted as a means of dealing with the swelling or sepsis the operation is followed by an intractable hæmorrhage, which is one of the symptoms of the disease, and may prove fatal.

The diagnosis is decided by the blood count. There is a great excess, absolute and real, of large mononuclear non-granular cells, which may be either myeloblasts or lymphoblasts. The prognosis is bad, in some cases death occurring within a few weeks or months.

It is believed now that infection from the mouth has nothing to do with the etiology of this disease, though infection in the mouth may decide the incidence and site of the oral lesion.

It is of importance, however, for the dental surgeon to consider systemic conditions in cases of acute dental sepsis before undertaking extractions for its cure, as in this disease the risk of a fatal termination is greatly increased if they are undertaken.

In a case in which with repeated blood transfusions the patient lived for two and a half months, the course taken by the initial oral lesions was as follows: beneath the swellings there was superficial necrosis of the alveolar bone, and the teeth there losing their support were cast off; as the sequestra separated and came away healing began to take place slowly.

(G) SCURVY.

When the gums are affected by scurvy they become dark, turgid, spongy, and so swollen as to hide a considerable portion of the surfaces of the teeth. At first the gums bleed readily; this is followed later on by a constant oozing. Ulceration and sloughing of the edges of the gums take place, leading to loosening and loss of the teeth and necrosis of portions of the jaws. In scurvy, the mucous membranes are anæmic, and there will be a strong contrast between the reddened gum and the pale mucous membrane covering the lips. Petechiæ or purpuric patches may occur on the hard and soft palate and on the limbs, particularly on the extensor aspects of the lower limbs, and joints may become distended with fluid. W. H. Willcox states[1] that 'a characteristic dark crimson or purplish area of discoloration commonly extends from the inner aspect of the molar teeth upwards towards the palate.' The local symptoms are nearly always associated with marked general symptoms indicative of the disease. In infantile scurvy changes in the gums are absent if no teeth have erupted, and in scorbutic adults the gums show no change if the patients are edentulous.

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[1] *Proc. Roy. Soc. Med.* (Section of Therapeutics), xiii, 78.

Scurvy is a rare disease in England, but it is possible that mild degrees of sub-scurvy are common, and this should always be considered when a gingivitis for which no satisfactory explanation can be found is encountered.

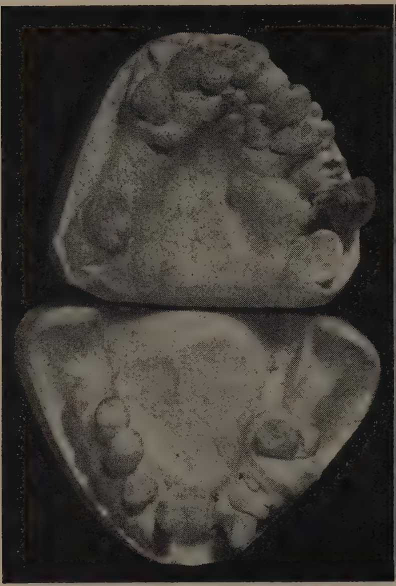

Fig. 574.—Scurvy (English), male aged 71. (E. S.)

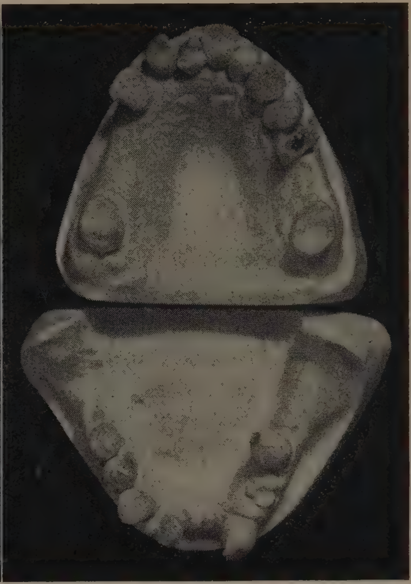

Fig. 575.—Same as Fig. 574 after about three weeks' treatment with ascorbic acid. (E. S.)

It does occasionally occur in this country. Figs. 574 and 575 are the models of a male old age pensioner aged seventy-one, who had never been out of England and who had scurvy; his diet consisted mainly of buns and tea. Fig. 575 shows the change in his oral condition after three weeks' treatment with ascorbic acid. Most present-day scurvy in England occurs in elderly males who are either bachelors or widowers.

Scurvy is a disease due to deficiency of vitamin C, and its treatment consists in the giving of such foods as contain abundance of vitamin C, as oranges, lemon juice, tomatoes, and uncooked cabbage, or the administration of the pure chemical vitamin as ascorbic acid, any excess being excreted in the urine. The mouth must be kept clean by the frequent use of antiseptic lotions and any radical treatment avoided as far as possible for the reasons given under 'purpura.' The condition of the gums has been likened to multiple epulides.

According to Budd[1]: 'The efficacy of oranges in preventing and curing scurvy was discovered before the disease had been described by the physicians.' Ronsseus, one of the earliest writers on scurvy, in a work published in 1564, observed that seamen on long voyages cured themselves of it by the use of oranges, and stated: 'this property was discovered accidentally and by chance by some Dutch sailors returning from Spain who were attracted by the novel richness of oranges, and by their greedy gluttony unexpectedly drove out the disease and had this happy experience not once but repeatedly.'

From 1601 onwards, on the advice of Sir James Lancaster, the East India Company's ships carried a supply of oranges and lemons, or lemon juice, for the prevention of scurvy among their crews. In Captain Cook's voyage in 1769 the Royal Navy took to this means of preventing scurvy, but it was not formally adopted by the Admiralty for this purpose till 1795. 'Even then it was misunderstood, and very often the juice of West Indian limes was provided with disastrous results. The story of Scott's voyage to the Antarctic, and of the Mesopotamian Expeditionary Force demonstrate how long it was before the value of Sir James Lancaster's experience and the practice of the East India Company was fully appreciated.'

(H) PERFORATING ULCERS OF THE MOUTH.

Such ulcers, which are due to trophic changes, are occasionally seen in patients who are the subjects of *tabes dorsalis*.

As to the explanation of the lesion, there are two views: (1) that of Galippe, that it is the result of parodontal disease aggravated by tabes; and (2) that of Baudet, that it is an atrophic tabetic lesion, due to the fifth nerve being affected, which begins by loss of the teeth and atrophy of the jaws, and sometimes terminates by ulceration and perforation into the maxillary sinus.

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[1] The historical details are extracted from a paper by J. A. Nixon, *Proc. Roy. Soc. Med.*, 1938, xxxi, 193.

(I) TUBERCLE.

Tuberculosis of the gums or mucous membrane of the mouth is a rare condition and seldom arises as a primary lesion.

The changes in the mucous membrane are similar to those seen when the disease is located in the skin. The tubercular nodules are present as minute white points set in a red thickened membrane. Ulceration occurs at an early stage owing to 'the constant maceration of the newly formed abnormal tissues by the fluids of the mouth.' The ulcerating patch has well-defined borders. 'The most characteristic feature of tubercular ulcers of the mucous membrane of the mouth is the presence of minute tubercular nodules in the margins and underneath the layer of granulations.' The colour of the granulations present in tubercular ulcers has aptly been likened to that of apple jelly.

A. Pain [1] records the case of a female, aged twenty-four, in whom the typical signs

> 'extended laterally along the labial surface of the upper jaw from the midline to the first premolar on each side, and vertically from the necks of the teeth to the sulcus where the mucous membrane was reflected on to the lip. There was also a patch in the middle of the hard palate about the size of a sixpenny piece. In appearance the gums were red, spongy, slightly raised, with an uneven granular surface upon which minute ulcers were present, and when pressure was applied with a glass slide the typical apple jelly nodules could be seen. The patient gave the history that the trouble had started seven years before with a redness of the gums round the edges of the maxillary first incisors.'

When she was sent for light treatment pulmonary tuberculosis was found to be present.

(J) SYPHILITIC INFLAMMATION AND ULCERATION.

Syphilis may affect the gums and the oral mucous membrane in any of its stages.

The primary lesion may occur on the gums, tongue, or other parts, and often assumes an unusual appearance. Ulcers of peculiar appearance and uncertain origin should always be regarded with suspicion, and the possibility that they are of syphilitic origin should be carefully inquired into.

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[1] *Brit. Dent. Journ.*, lviii. 23.

Secondary lesions may appear in the form of mucous patches in almost any part of the mouth ; the favourite situations are the inner surfaces of the cheeks, the edges of the tongue and the angles of the mouth. In weak and debilitated patients, the mucous patch may break down, leaving an ulcer with a sinuous outline. The ulceration may extend and lead to extensive destruction of the tissue, followed, on healing, by adhesion of contiguous parts and extensive scar contraction.

In the tertiary stages the ulcerations are generally of a deep excavated character, and are preceded by gummata, which, undergoing degeneration, produce the ulceration.

It is of great importance that before commencement of a course of anti-syphilitic treatment the patient's mouth should be put into as functional and non-infective a state as possible, and in undertaking such work the dental surgeon should wear gloves.

Bejel.—This is the name given to a non-venereal form of syphilis commonly found among Bedouin nomads. According to Hudson [1] 75 per cent. adults say they have had the disease, 60 per cent. have positive blood reactions, and surveys based on serology, history, and physical examination have led to the conclusion that 90 per cent. of the adult population are affected.

The disease appears to be transmitted chiefly in childhood by immediate non-sexual contacts, but no primary sore has been identified with it.

The lesions on the mucous membranes of the mouth are those of secondary syphilis, and spirochætes are readily demonstrable in both mucous and cutaneous lesions. These disappear spontaneously, and after a latent period, which may be many years in length, later lesions occur. The commonest is an ulcer involving any or all of the structures in the oral, nasal or pharyngeal cavities ; these lesions are gummatous in nature and spirochætes are not usually found in them ; trauma seems sometimes to determine their site.

The characteristic ulcer of late bejel begins with a painful congestion which ulcerates, the ulcer extending to a large size and being covered with a yellow slough. The lesions readily respond to arsenical and bismuth anti-syphilitic treatment, but if left untreated heal slowly with extensive formation of scar tissue.

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[1] *The Dental Magazine*, 1937, liv, 1068.

(K) PERIARTERITIS NODOSA.

This disease may rarely give rise to oral manifestations. It is usually [1] regarded as a rare disease having the character of an infection or toxæmia, ending fatally within a few months, seldom recognised during life and usually diagnosed post mortem.

Its rarity is now believed to be partly, if not largely, due to unfamiliarity with the disease and its diagnosis, and as more is learnt of it, it is found to be not necessarily or even usually fatal.

There are no definite clinical manifestations of the disease, which may occur at any age and more often in males than in females in the proportion of 4 to 1; the cause is unknown, though some think it may be due to an unknown specific infection. 'A complete list of the symptoms of the established disease would cover almost the whole field of medicine.'

In some cases it proceeds to a fatal termination within a few weeks, though it has been known to continue with remissions and intermissions for as long as 32 years. The disease also occurs in several species of ungulates, and dogs, and in the axis deer appears to occur in epidemic form.

The chief diagnostic features in practice are local symptoms due to the inflammation of small arteries, and nodules along the course of superficial arteries; large arteries are seldom affected though small veins may be; any part of the body may be involved.

Microscopically the chief feature is a hyaline or fibrinoid necrosis of the arterial wall together with a surrounding inflammatory reaction.

The following is an account of the oral (only) signs, symptoms and microscopical findings in a case which was diagnosed post mortem:

> 'A. R., a clerk, aged 20 years, was admitted to hospital on October 16, 1939, on account of an inflammatory condition of the gums, fever and vomiting. He died ten days later, the total duration of his illness being between two and three months.
>
> 'Until recently the patient had enjoyed good general health. In August, 1939, while at a holiday camp, he noticed shortness of breath on exertion, on account of which he had to give up swimming; he began to lose weight. In the beginning of September he had a slight cold and noticed small raised areas on both upper and lower gums near the first molar tooth on the right side. On September 7, the lower right first molar tooth was extracted. The gums became

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[1] For the following details we are indebted to R. T. Grant. *Clinical Science*, 1940, 4, 245.

more generally inflamed and bled when he cleaned his teeth. He attended the Dental Department of this hospital (Guy's) on September 21. His temperature was then 98·8° F. The gums on the right side were inflamed as far as the mid-line. A swollen purplish area with a granular surface extended from the right lower canine to the right second molar; a similar smaller area was seen at the upper right first molar. A week later the condition had spread to the left gums; the jaw was not painful. His temperature was 97·8° F. and the blood pressure 130/85 mm. Hg. No other sign of disease was noted; the lymphatic glands were not enlarged, the spleen was not palpable; there was no purpura; the reflexes, fundi, pupils, and urine were normal. A blood smear showed no abnormality. A portion of inflamed gum was excised and examined microscopically; it showed septic granulation tissue covered with epithelium. By October 13, the gums had not improved; the patient complained of aching pains in his limbs, of nasal obstruction and bloody discharge, and of vomiting. His temperature was 100·2° F. He was admitted to hospital two days later for further investigation. At that time the temperature had risen to 101·5° F.; the pulse rate was 125 per min., and the blood pressure 120/75 mm. Hg. The gums were much inflamed, the mouth dry and dirty and the breath foul. The tonsils were enlarged. The nose was swollen and tender to the touch; its mucous membrane was hyperæmic and bled easily; the nasal cavities were blocked by firm blood clot. Another portion of inflamed gum was removed for microscopical examination.

'On October 24 further hæmorrhagic lesions appeared around the mouth. On October 25 he died.

'Microscopical examination of the two pieces of gum excised during life revealed a complex picture of acute and subacute inflammation. Only very small blood vessels were present. Many of these showed an acute necrosis with thrombosis, hæmorrhage into the surrounding tissue spaces, and dense polymorphonuclear infiltration. Other areas showed a subacute inflammatory reaction with commencing proliferation of fibroblasts. Many eosinophil leucocytes and foreign body giant cells were present.'

(L) ANEURISM.

A case is recorded by J. H. Edward[1] in which the anterior palatine artery entered the mouth through an aperture in the centre of the suture between the palatine bones. The pressure of a denture at this part led to an aneurismal dilation of the vessel.

The various tumours arising in connection with the gums are described in Chapter XXXII.

General Note.

It will be noticed in many varieties of stomatitis, notably such as ulcerative stomatitis, cancrum oris, scurvy, mercurial stomatitis, the leukæmias, and the necrosis which occurs occasionally in children suffering from acute infective fevers, that, quite apart from infection with any specific organism as a cause of the stomatitis, the resistance of the individual is first lowered (and often extremely so) by faulty environment, food deficiency, exposure, or the specific infection or other factors causing the general disease. Under these circumstances, any potentially pathogenic organism which inhabits the normal mouth may flourish and be enabled to damage the injured tissues, and it is probable that infection with a specific organism as a cause of any of the type of lesions enumerated is rare, but that the lowered resistance of the body lays it open to the attacks of whatever potentially pathogenic organisms may be present, and that the symptoms to which they give rise depend on the depth to which the resistance is lowered, the virulence of the organisms present, and the type or types which may be in preponderance. Obviously, the more clean (the less infected) the mouth is to begin with the more resistant will its tissues prove to the causative factors of such lesions; this is shown by the absence of these lesions in scurvy, mercurial stomatitis, etc., when there are no teeth present.

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[1] *Brit. Dent. Journ.*, xvi, 272.

(M) DISCOLORATIONS AND PIGMENTATIONS.

In addition to the discolorations of the gums and mucous membranes mentioned on pp. 510–513, 532 and 965, others occur which may occasionally be of value as aids in positive or negative diagnosis. These occur in:

Addison's Disease.—Brownish areas of pigmentation may occur on the gums and mucous membrane, and also on the lips, palate and tongue in this disease, and just as when they occur on the skin may be more noticeable or appear first in areas subjected to pressure such as may occur when dentures are worn; taken into consideration with a marked apparent sun-tan of the facial skin, anorexia and other signs and symptoms, this sign may be of considerable diagnostic value. The pigmentation is due to a deposit of melanin.

Bismuth Staining.—When bismuth is administered as an anti-syphilitic remedy there is usually a deposit of bismuth sulphide seen following the contour of the gingival margins around the teeth; this is particularly marked when any gingival sepsis is present; should there be any ulceration on the gums or mucous membranes these areas will also be outlined by a similar deposit. This deposit is usually described as bluish, and somewhat simulating Grisolle's line in chronic lead poisoning, but in the writer's experience it is generally rather browner, and sometimes indeed brown, and fairly readily differentiated. The deposit probably occurs in the same manner as the deposit of lead sulphide in chronic lead poisoning and in the absence of teeth does not occur.

Argyria.—Silver may gain access to the body in a variety of ways, usually as an occupational disability, but occasionally as a sequel to prolonged medical treatment with silver salts. It is in the skin that the slaty-grey discoloration is usually noticed, and in the majority of its methods of access the gums and mucous membrane do not seem to be affected, but in people engaged in cutting up silver-leaf into books discoloration of the gums and mucous membranes has been recorded.[1] Gum in contact with an amalgam filling containing silver may occasionally show a localised area of dark metallic discoloration.

Foreign Matter in the Tissues.—A case occurring in the practice of A. E. W. Miles was that of an edentulous patient having small bluish-black areas localised to a confined space of the mandibular gum ; on opening up one of these a small piece of amalgam was removed, though considerably smaller than the area of discoloration. It is presumed that when the extractions were done an amalgam filling containing a preponderance of silver was splintered, and the pieces left in the wound became enclosed in the tissues in the process of healing ; and that the discoloration was due to areas of deposit of silver sulphide having origin from each piece of amalgam and forming haloes of deposit around them.

Physiological Discoloration.—Negroid and other dark-skinned races show irregular brownish areas, chiefly on the labial and buccal aspects of the gums, and also on the palate and mucous membrane ; in hybrids between them and white races, such as mulattos, the brown coloration may also occur in patches ; sometimes the patches are large, but they may present a freckled appearance.

(N) NEOPLASMS.

Such neoplasms as may originate with or affect the gums are dealt with in Chapter XXXII.

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[1] F. Koelsch, *Münch. med. Woch.*, 1912, lix, 304.

CHAPTER XVII

An Introduction to Dental Radiology,

By the late Stanley Colyer, M.D. Lond., D.M.R.E.

'*Detail is Everything.*'

(A) INTRODUCTORY.

To understand the principles upon which radiology is based it is necessary to have an elementary idea of the structure of matter. No attempt will be made in this chapter to deal with the question in an extensive fashion, but it is felt that a few remarks which may help the student to get a working conception of a very difficult subject will not be out of place.

'We live,' says Sir James Jeans in one of his picturesque and illuminating phrases, 'in a gossamer universe; pattern, plan and design are there in abundance, but solid substance is rare.' So-called atoms are not indivisible particles of matter, but consist of a nucleus, called the proton, with one (as in the case of hydrogen), or more (as in the case of other elements), electrons whirling round the proton at incredible speed. The electrons are at comparatively enormous distances from the proton; thus, to take another sentence from Jeans, '. . . a few wasps—six for an atom of carbon—flying around in Waterloo station represent the extent to which the atom is crowded with electrons—all the rest is emptiness.'

Light and all other forms of radiation travel at the rate of 186,000 miles per second, the differences in radiation depending upon their wave-lengths. At one end of the visible spectrum are the wireless waves of hundreds of yards, and at the other end will be found those of the X-rays, with wave-lengths of about one hundredth to a thousandth of the visible rays. These X-rays are so short that they can pass through atoms. Now the atomic number of an atom is 'the number of electrons that fly round in orbits in an atom.' Thus the atomic number of hydrogen is one, of helium two, and of uranium, the heaviest metal known on earth, ninety-two, with a nucleus 238 times the weight of the hydrogen nucleus.

As the result of experiments Rutherford was forced to the conclusion that the chemical properties and the nature of the atom resided in the central nucleus which carries a positive charge of electricity which exactly neutralises the negative charge carried by the electrons. It is the nucleus of the atom which is supposed to interfere with the passage of the X-rays, and it has been found that the capacity of substances to stop the rays is in proportion to their atomic numbers. If the word density be substituted for atomic number, then the higher the density of a substance the more X-rays it will stop. An X-ray may pass through an atom, may hit a nucleus head-on and be stopped, or it may hit the side of a nucleus and be deviated. It follows from the above that the denser a substance the more opaque it will be to the X-rays, and in consequence its shadow, as shown on the photographic film, will be more complete and its edge will be sharper than the shadow of less dense substances. Dense bone, as sclerosed bone, will give dense shadows, more porous bone, as inflamed bone, will give less dense shadows with less defined edges, and soft tissue still less dense and definite shadows. Such differences are clearly shown in dental radiology in the differences between mature bone and immature bone, between normal and pathological bone.

(B) TECHNIQUE.

In order that radiology may be of real value to the science and practice of dentistry, it is essential that the radiographic work in relation to it shall be of a high order and shall be standardised, for without these two conditions comparisons, which are the basis of diagnosis, cannot safely be made. And it may be remarked here that the finer interpretation of dental radiograms is one of the more difficult branches of radiology.

It is not possible in a short textbook to go deeply into the subjects of technique and diagnosis, but they will be dealt with in sufficient detail to give the student a basis from which to work.

(1) Positioning the Patient.—The patient should sit upright so that his back, head and neck are vertical. The occlusal surface of the teeth to be radiographed should be horizontal.

To obtain this position it is necessary to *drop* the chin slightly in the case of the maxillary teeth, and to *raise* it slightly in the case of the mandibular.

Such a position may be obtained in an ordinary dental chair, but a special chair is preferable. As it is quicker when radiographing a whole mouth with small films to expose opposite films successively, either the patient or the apparatus should be capable of being moved to the corresponding sides. Unfortunately, the dental chair does not rotate round the axis of the spine of the patient, and it is for that reason that a special chair is an advantage. The special chair may be any chair with a straight back, such as a barber's chair, with a movable headrest attached. It is supported at three points only, of which one is fixed and two are movable. The fixed point consists of a vertical hollow iron tube supported on a bold base which rests on the floor, and into this is slipped a solid steel rod which is fixed above to the seat of the chair immediately under the spine of the patient. The vertical support is so arranged that it raises the back legs of the chair about half an inch from the floor. The two front supports consist of two rubber-tyred wire wheels, about eight inches in diameter, so fixed as to raise the front legs of the chair about half an inch from the floor. Across the front legs is fixed, about one inch from the floor, a board some six inches wide to receive the feet of the patient. The front wheels are so orientated that the chair can be rotated round the circumference of a circle of which the fixed point of the chair is the centre. It is clear that in such a chair the patient can be rotated so as to bring the corresponding parts of his jaws into similar relation to the X-ray tube.

(2) Positioning of the Film.—It is not possible to do standardised radiography without the aid of dental film-holders. Of the many varieties on the market only those designed by the writer, and described by him in the *British Journal of Radiology*, ix, 105, will be described. The idea of holding a dental film in a gutter formed between a block of hard radiolucent material and a metal plate was first put into practice by C. A. Clark, and the film-holders to be described are based on them but differ from them in many important details. The most essential difference, and, perhaps, the most original, is that these holders are designed not only to be steady in the mouth but, if used according to the technique advised, are so designed *that the film is pressed firmly against the part to be radiographed.*

In the technique to be described considerable use is made of one of the fingers of the patient, though he is rarely allowed to take an active part, so to speak, in the work—for to ask for the intelligent co-operation of the patient is to ask for trouble. All that is required of the patient is that he shall lend an inert first finger (or occasionally another finger), which is placed by the operator where it is wanted, and then shall bite upon it. By this means the operator has to hand a convenient wedge which he can place anywhere in the mouth.

Each film-holder consists essentially of :—

1. A film-plate against which the film is placed.
2. A fibre block, or blocks, held between the teeth.
3. A gutter formed between one of the fibre blocks and the film-plate, and into which the film is slipped and by which it is held.
4. A handle by means of which the holder and film are introduced into the mouth by the operator.

In addition, in the case of the holders for the maxillary and mandibular incisor teeth, there are wings, and in the case of the other holders there is a finger-plate. These additions supply areas against which pressure can be exerted in different ways, and by means of which the film is not only steadied *but is rotated towards the part to be radiographed.*

The following description of each film-holder will make the above remarks clear.

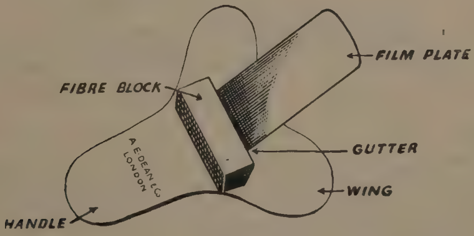

FIG. 576.

(1) *Holder for the Maxillary Incisors* (fig. 576).

The shape of the parts named above is clearly shown in the figure. The film is slipped well down into the gutter. If the patient has a broad palate the upper angles of the film are bent a little backwards so that its upper part may accommodate itself to the curve of the palate. If the patient has a narrower palate the sides of the film must be bent back along the sides of the film-plate. In the case of a very narrow palate the holder cannot be used, and use must be made of the thumb of the patient. To insert the holder the handle is held between the thumb and first finger of the operator, and his second finger is placed *behind* the film-plate. The holder is then introduced into the mouth, and by means of the second finger of the operator is pressed forward and upwards. The patient then closes his teeth upon the holder. This last movement brings the maxillary incisor teeth against the upper surface of the fibre block, and the mandibular incisors and canines against the wings. As the force exerted by the latter is behind the fulcrum, which is at the edge of the maxillary incisors, it will tend to rotate the film-plate against the palate behind the maxillary incisors.

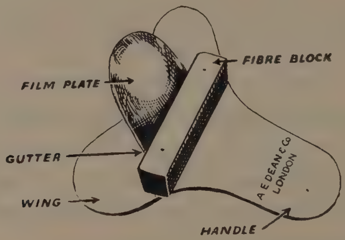

Fig. 577.

(2) *Holder for the Mandibular Incisors* (fig. 577).

The design and principles used in this holder are much the same as those used for the maxillary incisors, the main difference being that the film-plate is shorter, more vertical and slightly convex forward from side to side and from above downwards. The film is pressed well into the gutter and is bent round the film-plate more or less to the curve of the incisor region. The handle is held between the thumb and first finger of the operator, and as the holder is introduced into the mouth the handle is depressed a little below the level of the edge of the incisors. The fibre block is then placed firmly upon the edge of the incisors and the handle of the holder is slowly and firmly raised. This last movement rotates the film against the back of the bone of the incisor region and bends it to the curve of that part of the mandible. The patient then closes his jaws upon the holder.

This movement brings the maxillary incisors and canines against the wings, and as the pressure of the canines is behind the fulcrum which is at the edge of the mandibular incisors, the film-plate will be pressed against the back of the mandibular incisors.

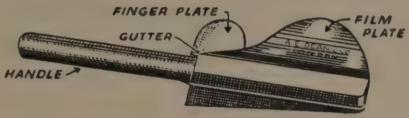

Fig. 578.

(3) *Holders for the Canines, Premolars, and Molars of both Upper and Lower Jaws* (figs. 578 and 579).

There are two holders which are similar, except that they are adapted to the opposite sides of the jaws. These holders, in addition to the fibre blocks, possess a small plate which passes inwards at right angles from between the fibre blocks and at their fore part. This small plate is called the *finger-plate.* Its use will be explained later. The fibre block away from the film side is wedge shaped, the apex of the wedge being backwards. This shape allows the fibre blocks to accommodate themselves, approximately, to the angular space which the maxillary and mandibular teeth make with each other when they are partly separated. When the film-holder is used for the mandible the fit is generally comparatively accurate, but when used for the maxillary teeth there is often left in the front a space which sometimes allows the film-holder to sag downwards. To overcome this trouble the first finger of the hand of the patient, *of the side opposite to the side being radiographed*, is taken by the operator, and the tip of it is placed under the finger-plate, and the patient is told to bite. The tip of the finger acts as a passive wedge, and its effect is not only to push up the upper fibre block against the occlusal surface of the maxillary teeth, but also to rotate the film against the side of the palate in relation to the teeth being radiographed. (It

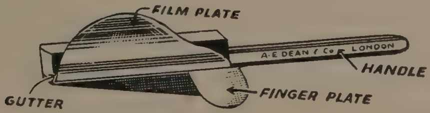

Fig. 579.

will be noticed that the patient is, practically speaking, a passive agent ; he merely lends the tip of his finger as a wedge, bites upon it and does nothing more. Sometimes it will be found more suitable to place the tip of the patient's finger upon the mandibular teeth below the lower fibre block, but generally the position below the finger-plate will be found the more convenient. In a few cases the holder fits satisfactorily and the use of the finger is not called for.)

A few additional points may be noted :—

(*a*) In radiographing the maxillary canines and first premolars the film is placed in the gutter *so that its back edge is in a line with the posterior ends* of the fibre blocks, and the antero-superior angle of the film is bent inwards so that it may accommodate itself more easily to the shape of the palate.

(*b*) In radiographing the maxillary premolars and molars the film is placed in the gutter *so that its front edge is in a line with the anterior ends of the fibre blocks*, and the postero-superior angle of the film is bent slightly inwards so that the film may accommodate itself more easily to the shape of the palate.

(*c*) In radiographing the mandibular canines and first premolars the film is placed forwards as in the case of the corresponding upper teeth, and the antero-inferior angle of the film is bent slightly inwards so that it may accommodate itself to the curve of the mandible.

(*d*) In radiographing the mandibular premolars and molars the film is placed backwards as in the case of the corresponding maxillary teeth, but it is not necessary to bend the film.

A few words are necessary as to the care of the holders. It is advisable once the film-holders have been wetted [1] they should not be allowed to dry again, otherwise the fibre blocks will warp and be ruined. Once wetted they should be kept permanently in a weak antiseptic solution, such as carbolic acid 1 in 200.

(3) On the Direction of the X-ray Beam.—There is no simple golden rule to guide the operator with reference to the relative positions of the film, the ray, and the teeth. He must rely upon his knowledge of anatomy and his own common sense. The object to be aimed at is to produce a shadow of an object which shall be of its same size and profile. There is one principle only as to the direction of the ray, and that is that it shall strike at right angles an imaginary plane situated at an equal distance from the film and from the object to be radiographed. As to produce such a condition the film must be flat, this is technically possible in the case of the maxillary first incisors only. The principle, however, is of great importance and will serve to illustrate the different effects produced of placing the film in different positions in reference to the tooth. If the dark thick line AB (fig. 580) represent the tooth, the line DB the position of the film, and CB an imaginary plane between the two which bisects the angle at which they meet, then in order that the shadow BD′ produced on the film may be equal to the length of the shadow AB the main ray must strike the imaginary plane at right angles. This is the principle of the isosceles triangle. If the film be moved into any position farther away from the imaginary plane and the direction of the principal ray remain constant, then the shadow of the line AB will be gradually lengthened as the film is moved farther and farther away.

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[1] The first effect of wetting the film-holders is to cause a swelling of the fibre blocks, which leads to a narrowing of the gutter and consequently to difficulty in inserting the film. It is practically impossible to guard against this trouble. To overcome it the operator should carefully enlarge the gutter a day or so after the holders have been soaked. This can be done with a thin knife or an old safety razor blade. Various substances in place of fibre have been tested, but none has proved as satisfactory. Fibre is not only fairly radiolucent, but it is sufficiently soft when wet to allow the cusps of the teeth to indent it, and thus to obtain a firm grip which ensures steadiness.

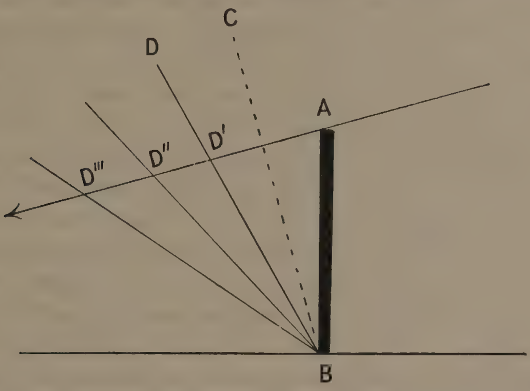

FIG. 580.—To illustrate the relation between the direction of the main X-ray beam, the film and the object to be radiographed.

AB = position of object.
BD = position of film.
BC = an imaginary plane bisecting AB and BD.
BD′ = length of shadow when in correct position.
BD″, BD‴ = lengthened shadow of object.

Contrariwise, if the film be moved nearer the plane the shadow of AB will be foreshortened. It follows from the above that although the film in dental radiography cannot be placed in the ideal position, the nearer it is to the tooth the more horizontal will be the direction of the ray, and the farther it is away the more vertical will be the angle. In practice the film is placed as near as possible to the tooth and the angle of the main ray is roughly calculated from the height and shape of the palate, which is ascertained by observation. Thus in the *maxilla* the higher the palate the more vertical will be the film and the more horizontal will be the angle of the principal ray. The most difficult cases are those with low palates, as not only has the angle of incidence to be more vertical, but the malar bone, which in them is often low and dense, has to be avoided. Means for overcoming this difficulty are discussed later, but it may be remarked here that it is sometimes insuperable. In the *mandible* the depth of the floor of the mouth will determine the angle of the X-ray beam. Thus, if the floor of the mouth is low the film will lie vertically, but if the floor of the mouth is high, then the lower part of the film will be curved medialwards. With the low, or deep, mouth the beam can be directed horizontally; with the high, or shallow, mouth the direction of the beam will be more vertical, the inclination towards the vertical being in direct relation to the medialwards curve of the lower part of the film.

(4) A Complete Examination of the Mouth.—A complete examination of the mouth can generally be made satisfactorily with ten films. The more quickly the work is done, owing to the sensitiveness of the mouth, the more likely is the result to be satisfactory. The difficulty in overcoming a sensitive mouth is in some cases very considerable. The following methods will each show good results in certain cases: (1) Slow, deep, regular breathing *through the mouth* while the film is in position. (2) By getting the patient to rub roughly the sensitive part with one of his fingers. (3) By dabbing the sensitive part with weak carbolic acid, about 1 in 40, or by swilling the mouth and holding the solution in the mouth for some time of a weaker solution, about 1 in 100 to 200. (4) By sucking a few tablets of 'Pastilles Rhodia'—a proprietary article. These tablets to be effective should be sucked for at least ten minutes before starting the operation.

The order in which the films are taken is immaterial. The direction of the X-ray tube should be arranged approximately before the insertion of the film (figs. 593 and 610 for positions).

Film No. 1. *Maxillary First Incisors* (and occasionally, in patients with broad palates, the second incisors).—The main ray is entered in a downward direction at the midpoint of the roots of the incisors and should strike an imaginary plane midway between the film and the teeth at right angles.

Films Nos. 2 *and* 3. *Second Incisors, Canines, and frequently the First Premolars.*—The film should be so placed in the mouth that its anterior edge bisects the lower border of the first incisor. The main ray is directed downwards and strikes the canine at about the junction of its upper and middle thirds.

Films Nos. 4 *and* 5. *Premolars and Molars.*—In the absence of the third molars an ordinary dental film will cover these teeth. If the third molars are present it may be necessary to take two films, one of which can be discarded later if a good shadow of the first premolar is found on the canine film. The direction of the ray varies with the position of the malar bone (fig. 581). As a rule the ray is directed downwards, inwards, and a little backwards, so as just to miss the lower edge of the malar bone and the arched lower border of zygomatic process of the maxilla. In difficult cases with a low malar bone it may be necessary to radiograph through that bone, which means that clear shadows of the molar roots will not be obtained. In certain cases clear shadows of the second and third molars may be obtained by entering the rays downwards and a little forwards behind the arched lower border of the zygomatic process of the maxilla.

Film No. 6. *Mandibular Incisors.*—The direction of the ray is upwards. In many cases a considerable inclination is necessary. This inclination has to be increased in proportion to the curve backwards of the film brought about by the floor of the mouth.

Films Nos. 7 *and* 8. *Mandibular Canines and First Premolars.*—The anterior edge of the film should not quite reach to the medial edge of the second incisor. The direction of the ray should be directly over the root of the canine and have a well-marked inclination upwards. Its exact position will depend upon the position of the canine in relation to the curve of the mandible and to the depth of the floor of the mouth.

Films Nos. 9 *and* 10. *Mandibular Premolars and Molars.*—

The film, as a rule, can be made to cover four of these teeth. The same remarks apply here with regard to the presence of the third molars as to the corresponding maxillary teeth. The main ray should be entered horizontally, or with a slight inclination upwards.

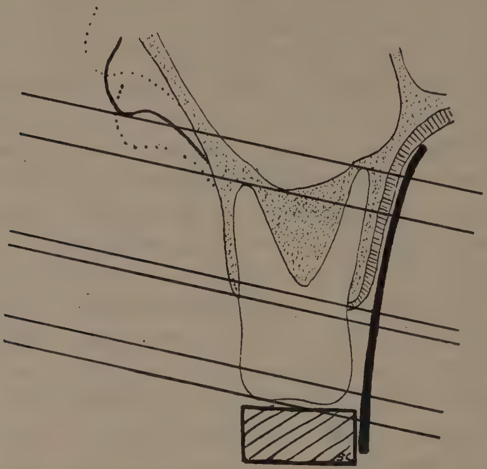

FIG. 581.—To illustrate difficulties in radiographing the maxillary molars. The diagram is drawn from a bony specimen through the region of M. 1. The malar process of the maxilla and its curved lower border is approximately opposite the junction between M. 1 and M. 2.

Note the following points :—

1. The main X-ray beam should just miss the malar bone.
2. If the rays can strike the film more or less at right angles then the palatine and buccal roots will be of approximately normal length.
3. If the palate is high the direction of the rays should be lowered, and would give a still more correct shadow of the roots.
4. If the palate is low it is impossible to raise the direction of the rays without the intervention of the malar bone. If the direction is not raised the shadow of the palatine root will be lengthened. If the direction is raised the shadow of the palatine root may be normal, but it will be obscured by the shadow of the malar bone.
5. The higher the rays are raised the shorter will become the shadow of the buccal roots.
6. The shadow of the palatine root is always seen through the floor of the antrum.
7. The shadows of the buccal roots will overlap and partly merge into that of the palatine root.
8. The shadow of the edge of the buccal side of the alveolar process will be lower than the edge of the lingual side.

The technique used in producing radiograms of the teeth, as described above, brings about certain distortions in their shadows. These are :—

(*a*) *In the Maxilla.*—The first and second incisors are of approximately correct length. The premolars on the film which shows a comparatively correct length of a canine will be foreshortened, whilst on the molar film they will be about correct. The molars—the palatine root is seen through the floor of the antrum and is lengthened. The buccal root shadows are shortened and often merge into the shadow of the body of the tooth.

(*b*) *In the Mandible.*—The incisors may be a little foreshortened. The premolars on the canine film are foreshortened, whilst the canine root should be of approximately correct length. The premolars and molars on the molar film should be of correct length.

(5) Occlusal Films.—Occlusal films, taken either inferosuperiorly or supero-inferiorly, often afford information that cannot be obtained as satisfactorily in any other way. The logical alternative to their use is stereoscopic films, but experience does not justify their frequent use.

Occlusal films are of three sizes: large, medium, and small, the last being the ordinary dental film. The only principle that has to be observed in occlusal radiography is that the main ray shall as far as possible be at right angles to the film. The film is placed between the teeth and the patient is told to close his teeth upon it. This operation is likely to distort the film. This disadvantage is easily overcome by placing a metal plate, cut rather smaller than the film, below in the case of a supero-inferior film and above in the case of an infero-superior. The following points may be observed :—

(1) *Supero-inferior Film of the whole Maxilla.*—The head of the patient is placed vertically and the film is inserted into the mouth as far back as possible. The main ray is entered from above through the base of the nose. Depending upon the lie of the frontal bone in relation to the jaws, it may or may not be found necessary to give a tilt upwards to the head so as to prevent the shadow of the frontal bone being thrown over the shadow of the posterior teeth. The tilting of the chin is often of advantage when a radiogram of unerupted maxillary third molars is required, as these teeth generally lie looking a little backwards and outwards. The ray, under these circumstances, will frequently pass through the length of the teeth.

(2) *Infero-superior of the whole Mandible.*—The patient

should be placed as horizontal as possible, and the mandible should be tilted well upwards. The main ray is entered about one inch behind the symphysis in the mid-line.

(3) *Occlusal Films of Small Areas.*—When radiograms of small areas only are required the ordinary dental film may be used. The head of the patient is tilted into a suitable position. The main ray is entered immediately above or below the film as the case may be.

A convenient position for obtaining infero-superior views of the mandibular third molars may be noted here. A small film is placed over the probable position of the tooth, the mouth closed. The head is then bent backwards and to the opposite side and is given *also a slight rotation to the same side.* The main ray is entered from below and at right angles to the film. If the shoulder of the patient is found to be in the way of the X-ray beam the hand and forearm of the side near the X-ray tube is placed behind the patient or behind the chair. This movement has the effect of dropping the shoulder.

(6) Extra-oral Films.—For most dental purposes the half-plate film is sufficiently large. It is placed between two intensifying films to increase the rapidity of the exposure, and is lodged in a suitable casette. The casette should be placed against the side of the jaws, the chin given a moderate tilt upwards and the head turned a little towards the side to be radiographed. These movements will open up the space between the spine and the angle of the jaw of the side opposite to that to be radiographed. The main ray is entered through the centre of the space so created, and is pointed so as to strike the area it is wished to radiograph. Care must be taken that the shadow of the jaw of the opposite side—that is the side near to the X-ray apparatus—does not cast a shadow over the particular area to be radiographed; and that the back of the side of the jaw being radiographed does not pass behind the level of the spine. The main ray hits the film obliquely and hence some distortion of the shadow is to be expected.

(7) Localising. (1) *Localising Unerupted Teeth, or Roots, in Relation to Erupted Teeth.*—Ordinary lateral films are taken, and these are followed by a supero-inferior, or an infero-superior, as the case requires. By these means we get the horizontal and vertical lie of the unerupted tooth, or root, and from the films measurements can be taken in each direction, and thus the position of the unerupted tooth, or root, in relation to the erupted teeth can be calculated.

(2) *Unerupted Teeth, or Roots, in Edentulous Areas.*—The simplest method is to make a rough dental plate of wax or other suitable material, and to incorporate in it a few bits of thin iron wire, or a flattened-out coil of thin iron wire. The plate is left in position when the lateral and occlusal films are taken. The relation of the unerupted tooth, or root, to certain pieces of wire, or to certain coils of the wire, is thus obtained. The plate is then replaced in the mouth and the position noted on the plate is marked on the gum.

(3) **Fractures of the Jaws.** (*a*) *Of the Maxilla.*—Fractures of the maxilla are, as a rule, best radiographed by means of intra-oral films. Lateral and supero-inferior films should be taken.

(*b*) *Of the Mandible.*—Fractures of the mandible are often accompanied by pain and by marked displacement of the fragments, and the taking of intra-oral films may thus be rendered very difficult or even impossible. It is therefore generally necessary to take extra-oral films, and, in order that the position of the fragments may be accurately gauged, it is advisable to take stereoscopic films. In taking stereoscopic films provision must be made for changing the casette without moving the patient. The casette is placed in position and the head of the patient is placed against it in the position described for taking lateral views of the jaws. The X-ray tube is removed to, say, 20 inches from the film, and the main ray is directed upon the part. The position is noted, and the tube is then shifted lateralwards for, say, 1½ inches. The exposure is made, the casette is changed and the tube is shifted 3 inches to the opposite side, and a second exposure made. It is necessary to examine the films on a stereoscope.

(8) Exposure Technique.—As each apparatus varies the time of exposure for films can be given approximately only. For radiotised dental films, working at about 40 cm. (16 inches) at 60–65 kv., and 20 ma., about four to seven seconds will be found to be necessary, the time of exposure depending upon the thickness and density of the part to be radiographed. For extra-oral work, working at from 50–60 cm. (20–24 inches), at 50 kv. and 20 ma., give about three seconds.

(9) The Temporo-mandibular Articulation.—This is usually a difficult radiogram to obtain clearly owing to the relation of the articulation to the base of the skull, and it will often be found necessary to take several before the optimum position is obtained. Of the various methods the film taken with the beam directed from above gives the most satisfactory and least distorted view. The following is the method: Place the joint to be examined against the centre of the casette so that the zygoma is horizontal. Turn the face very slightly towards the film so that a line at right angles to the centre of the plate will pass through the joint and external auditory meatus of the opposite side. The central ray should enter the skull about 4 cm. above the external auditory meatus of the side opposite to the joint being radiographed, and it should emerge through the joint itself. The tilt of the tube is about 15 degrees. Distance about 50 cm. Exposure about 3 seconds at 65 kv. and 20 ma. (fig. 582, see plate A facing p. 580.)

(10) Examination of the Antra.—It is often necessary to examine the antra for suppuration, or for the presence of a foreign body. The diagnosis of antral suppuration may be made with intra-oral films, or extra-oral, the latter being the more usual method.

Intra-oral Films.—In order to make a reasonably safe diagnosis with intra-oral films it is necessary that the technique shall be standardised, or, in other words, that the corresponding films of the opposite side of the jaws shall be taken in the same position. If this is done it is possible to make a comparison of the two antral shadows.

Extra-oral Films.—This is the usual method. As the ray is entered from the back of the head the radiogram is said to give a postero-anterior view. Of the various positions the following one is, perhaps, the most satisfactory: A whole plate film enclosed in a casette is placed vertically upon a suitable stand. The face of the patient, who is sitting upright in a chair, is so placed that his chin just touches the bottom of the casette, and his head is then bent backwards until his nose is about $\frac{1}{2}$–$\frac{3}{4}$″ away from the casette. The ray is entered from behind so as to pass between the antra about their mid-point and strikes the film at right angles. If the position be correct the shadow of the petrous bones should be seen immediately below the lower angles of the shadows of the antral cavities. Distance about 26 inches. Exposure 5 seconds at 70 kv. and 20 ma. (Fig. 592, see plate B facing plate I.)

(11) The Mounting of Dental Films.—Intra-oral films should be mounted preferably upon transparent frosted celluloid paper, known as ‘Radio Paper.’ Small tongues, which serve to hold the films in position, can be cut by means of a carving tool of suitable shape. If a pattern of the position in which it is desired to mount the films be drawn in pencil on a sheet of paper, and this be placed under the transparent mount, it will show through, and the places at which it is required to cut the tongues will be seen. By this means films can be arranged in any position desired and a film mount can be cut in a few moments. From the point of view of interpretation it is desirable that the films be mounted close together.

(12) Systematic Examination of a Complete Set of Dental Radiograms.—Films in which fine detail is to be looked for should be examined in a darkened room against a strong transmitted light. It is asking the impossible to expect to see fine detail in any other way.

The films should first be viewed as a whole, and any change which is common to all—such, for example, as an increased or decreased brilliancy, an increased or a decreased thickness of trabeculæ, loss of alveolar edge, the presence of granulomata, the presence of rarefaction spaces, and so on—should be noted. Then a magnifying glass, of not less than two inches in diameter, should be taken, and commencing at the right upper molar region each tooth and its parodontal tissues should be examined in order. The condition of the alveolar edge, the periodontal membrane, the lamina dura, of the related interdental septa and of the bone deep to the apex of the tooth should be investigated. Systematically the examination of each film is made and when completed the general condition of the jaws and teeth is summed up. The radiographic evidence should then be checked against the clinical findings, and later against the pathological and post-operative findings. It is impressed upon the student that care and patience in examination are necessary, and only by close attention to detail can he hope to become a competent radiologist. It is not inapt to quote in this connection some words of Browning :—

Oh, the little more, and how much it is !
And the little less, and what worlds away !

(C) THE INTERPRETATION OF DENTAL RADIOGRAMS.

The interpretation of dental films, other than the localising of buried roots and teeth, and of fractures of the jaws, which have already been dealt with, falls into two groups: I. The interpretation of normal radiographic anatomy, and II. The interpretation of morbid radiographic anatomy, the latter of which may again be divided into changes of a non-inflammatory character including hyperplasia, and changes of an inflammatory character.

I. Normal Radiographic Anatomy.

Radiograms are composite shadows of solid tissues, or in other words, a radiogram is a two-dimensioned picture of a three-dimensioned tissue. They are unique: that is to say, whether made from normal or pathological tissue, they possess a character of their own apart from macroscopic or microscopic findings. To understand the abnormal it is necessary to study first the normal, and for the purposes of this article the normal is defined as a tissue which does not show evidence of the results of infection. Thus defined it will be found that the normal varies considerably, the majority of the variations being attributable to congenital, nutritional, and possibly, in some cases, to endocrine disturbances.

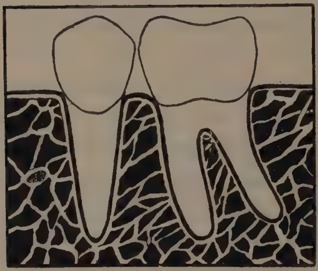

Fig. 583.—The *Periodontal Membrane Shadow* is the black line immediately outside the root of the teeth. The *laminal dural shadow* is the white line immediately to the outer side of this. Note the way the trabeculæ join it and join each other. Note in front of the root of the premolar a nutrient canal.

The following details in dental radiograms (negatives) should be noted (fig. 583):—

(1) The Periodontal Membrane.—The shadow of this tissue lies between that of the cancellous tissue and that of the root. It is a narrow black line which, for practical purposes, is of uniform thickness. When not deeply placed it is distinct, but when it is deeply placed, as about the apices of certain teeth, its clearness is interfered with by the shadows of the overlying tissues. At the cervical edge it becomes continuous with the shadows of the submucous tissues which show but faintly.

(2) The Lamina Dura.—The shadow of the lamina dura is sometimes known as the white line—the linea alba. It shows as a clear white line lining the sockets of all the teeth and is continuous with a similar white line covering the tops of the interdental septa. On its dental side it is normally smooth. On its cancellous side it is irregular and is continuous with that tissue to which it is joined by normally set cancellous trabeculæ. Actually the lamina dura is a rather dense cribriform layer of dense bone whose normal radiographic shadow is an unbroken line.

(3) The Cancellous Tissue.—Normal cancellous tissue can only be defined by a recognition of certain of its attributes in the undiseased state. The following description does not include abnormalities of structure due to early nutritional and, possibly, endocrine disturbances.

A close examination of cancellous tissue will discover the following peculiarities :—

(1) That an individual trabecula is not of uniform density, and that it has a finely granular character. The first of these peculiarities is due to alterations of its shape and position, the second to its porous structure.

(2) That trabeculæ meet each other at sharp—not rounded—angles, which angles may be acute or obtuse. From this it will follow that trabecular spaces will be irregular in outline, and that these irregularities will be sharply defined.

Here and there will be found vascular canals. The shadows of these canals are roundish or ovalish in shape. Their walls are of the character of trabeculæ, and the trabeculæ that support them meet them at sharp angles. The importance of this description of normal cancellous tissue will be appreciated when the changes due to chronic infection are described.

Less important characters to be noted are the differences to be seen in the pattern of the tissue at different places. The shape and position of trabeculæ in adult tissue are dependent upon the strains and stresses they are called upon to bear. In the *maxilla* the pattern is that of a finely meshed honeycomb with angular cells. In the *mandible*, which is of the nature of a slung arch, the pattern varies. Thus at the sides of the body the trabeculæ are coarse, and the cancellous spaces are large, the general trend of both being in a downward and forward direction, whilst in the incisor region, where the pressure is mainly vertical, the pattern of the structure approaches that of the maxilla.

(4) The Foramina.—The characters of the shadows of a foramen are that it is surrounded by bone of a finely granular structure, and that the trabeculæ which support it meet its wall at sharp angles—it thus shows similar characters to those of a nutrient canal, from which it differs in size only. The important foramina likely to be seen on dental films are :—

(*a*) *The Incisive Foramen* (*the Anterior Palatine*).—It is seen normally between the maxillary incisors. In size it varies within wide limits.

(*b*) *The Greater and Lesser Palatine Foramina* (*the Posterior Palatine Foramina*).—These are seen occasionally in films of the maxillary molar region. They do not add materially to the difficulties of interpretation.

(*c*) *The Mental Foramen.*—This is seen normally between and rather below the apices of the first and second mandibular premolars. It shows, as a rule, in addition to the characters mentioned above, shadows of trabeculæ crossing its shadow. These are shadows of the trabeculæ situated on the lingual side of the mandible.

(5) The Coronoid Process.—The tip of this is often seen at the back of maxillary molar films.

(6) The Maxillary Sinuses (the Antra).—The shadow of the anterior part of these sinuses is seen on the canine and molar films. The shadow of the lower border is seen in close relation to the apices of many teeth. The palatine roots of the molars are seen through the shadow of the floor of the sinus. Anteriorly the shadow of the lateral border of the antrum joins the shadow of the anterior wall which in its upper extremity joins the shadow of the floor of the nose. The antrum is very variable in size ; its anterior extremity lying anywhere between the plane of the first molar and that of the canine. The antral space may be divided by partitions, and the shadow of these may be mistaken for the walls of a multilocular cyst. In a backward direction the shadow of the floor of the antrum merges into the dense shadow of the malar bone.

(7) The Compact Bone.—The only compact bone of importance met with in dental films is that of the body and ramus of the mandible. Radiographically it is practically structureless, but shows a faint lineation in the direction of its length.

(8) The Teeth.—The enamel can always be distinguished from the dentine and occasionally the dentine can be distinguished from the cementum. As a rule the density of the root shadows can ‘absorb’ the shadows of the adjacent tissue without disturbing its own uniformity, but in the case of deeply placed roots in relation to coarse trabeculæ, such as the mandibular premolars, the shadows of the trabeculæ are seen superimposed upon the shadow of the roots : in them it is a normal and not a pathological condition.

(9) Changes in Radiographic Appearances due to Age.—The preceding description applies to normal adult tissue. In youth the tissues are moving onward towards the adult state. This immature tissue is usually clear, but lacks the sharp definition of good adult bone. The periodontal membranes are thicker, as are the laminæ duræ, and the arrangement of the trabeculæ is disturbed by the presence of unerupted teeth. The maturation of this tissue may be delayed so that in the early twenties it may sometimes be seen. This immature tissue in some respects mimics the appearances seen in early infection, and at times may present surprisingly difficult problems to the radiologist. In old age regressive changes are present. Radiograms show loss of lime salts in the bone, but not apparently in the teeth. The trabeculæ diminish in size, while the cancellous spaces show an increase. Somewhat similar changes may be seen in films of parts suffering from disuse.

II. MORBID RADIOGRAPHIC ANATOMY.

(1) Non-inflammatory Changes.—Under this heading come cysts, malignant and non-malignant neoplasms, developmental defects and hyperplasia. Though it is not proposed here to deal with these subjects in a lengthy fashion, as the descriptions and diagnoses are dealt with elsewhere in this book, it is felt that a few remarks from a radiologist’s point of view may be of some value to the student.

(*a*) *Cysts.*—These as a rule show as clear rounded translucent areas in the substance of the bone, and around the areas the bone adjacent to its wall shows some condensation (see fig. 602). If the cyst is uninfected the bone adjacent to its wall does not show infective changes (*q.v.*). If a cyst is multilocular it may show many rounded translucent areas separated from each other by rather denser linear shadows, which may be faint or well-marked, depending upon the thickness of the walls and the presence or absence of lime in them.

(*b*) *Endosteal Non-malignant Growths.*—These generally show as comparatively translucent areas of a somewhat irregular outline. Endosteal myelomata having lobulated surfaces, the irregularities of which fit into corresponding depressions in the bone wall, may give the appearance of having trabeculæ crossing the translucent area. The shadows simulating trabeculæ are the shadows of the edges of the bony depressions. The bone in relation to such a tumour may show some condensation, but it does not show inflammatory changes.

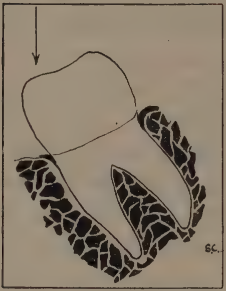

FIG. 584.—Compensatory hyperplasia. Note (1) that the periodontal membrane is not thickened; (2) that the trabeculæ meet the much thickened lamina dura at normal angles and that they are not thickened except where they join it.

(*c*) *Malignant Tumours.*—Except in extremely rare cases malignant tumours invade the bony tissues of the jaws from the outside, eroding the bone in relation to their advancing surface. The attacked bone seldom shows any reparative or infective changes.

(*d*) *Salivary Calculi.*—Calculi of the submaxillary glands are best shown on occlusal films. The calculi show as opaque areas not closely related to bone structures. They may be diagnosed from calcifying tumours of the floor of the mouth by the fact that the edge of the calcified area in the tumour is not as a rule sharp, and does not reach to the edge of the shadow representing the tumour.

(*e*) *Hyperplasia of Bone.*—Localised patches of hyperplasic bone occur in relation to misplaced teeth which are called upon to bear abnormal stresses and strains. The change is frequently seen in front of the anterior root of a mandibular third molar which has tilted forward and which has to support in its unnatural position an abnormal strain during mastication. Fig. 584 shows, diagrammatically, the changes which are found. The bone in the position of the lamina dura is thickened and the trabeculæ which join it show some thickening, but do not show the usual signs of inflammatory changes. The periodontal membrane is not thickened.

(2) Inflammatory Changes.—*A General Account of Infective Changes in the Bones.*—Chronic infection in, or in relation to, the bones of the jaws brings about reactive changes which it is the business of the radiologist to understand and interpret. The changes seen are unquestionable, and that certain of these changes are often associated with sickness in the patient is also unquestionable; but it is by no means certain how far these changes are due to the presence of organisms in the bone and how far they are due to toxins formed by organisms situated in and upon tissues about the bone and closely related to it. It is maintained by E. W. Fish[1] that all gum margins that have been examined, whether from the young or the old, show evidence of round-celled infiltration, and as I. Maclean admits that streptococci are demonstrable at a depth of two millimetres, it follows that we have an infected mesoblastic tissue in immediate contact with and morphologically related to bone. It is from a laboratory point of view a rather moot point whether these germs remain in the superficial tissues, or do pass from thence to the deeper tissues. Radiology certainly supports the second view, but it also supports the view that the main infection, but not necessarily the more toxic, remains localised to the surface tissues and that the deeper changes are to a large extent of a toxic character. But whatever may be the cause of the deeper tissue changes there is abundant evidence to show that the cause does not remain of a localised character but affects tissues over large areas. And further, that patients showing the more extensive changes of a certain character are those which appear to be suffering from oral infection. In assessing the importance of these deeper changes it is as well to remember that not only the germs but the damaged tissue itself may be, and probably is, toxic to the patient. When reading the following descriptions it should be remembered that in the matter of the explanation of inflammatory bone changes the radiologist led the way by many years, and that he had to rely mainly upon clinical observations. It may be that in the future he will, as the result of laboratory observations, be called upon to modify his views, but until such investigations are more advanced and have received general acceptance that time will not have arrived. Whatever may be the next advance in interpretation it will not alter the fact that the changes seen in bone give a measure of the extent and virulence of an infection about the jaws, and a measure of the resistance of the patient to that infection. For the present, therefore, the description of the changes seen in bone are based upon the assumption that they are due partly to toxins affecting the tissues and partly to germs infecting the bone.

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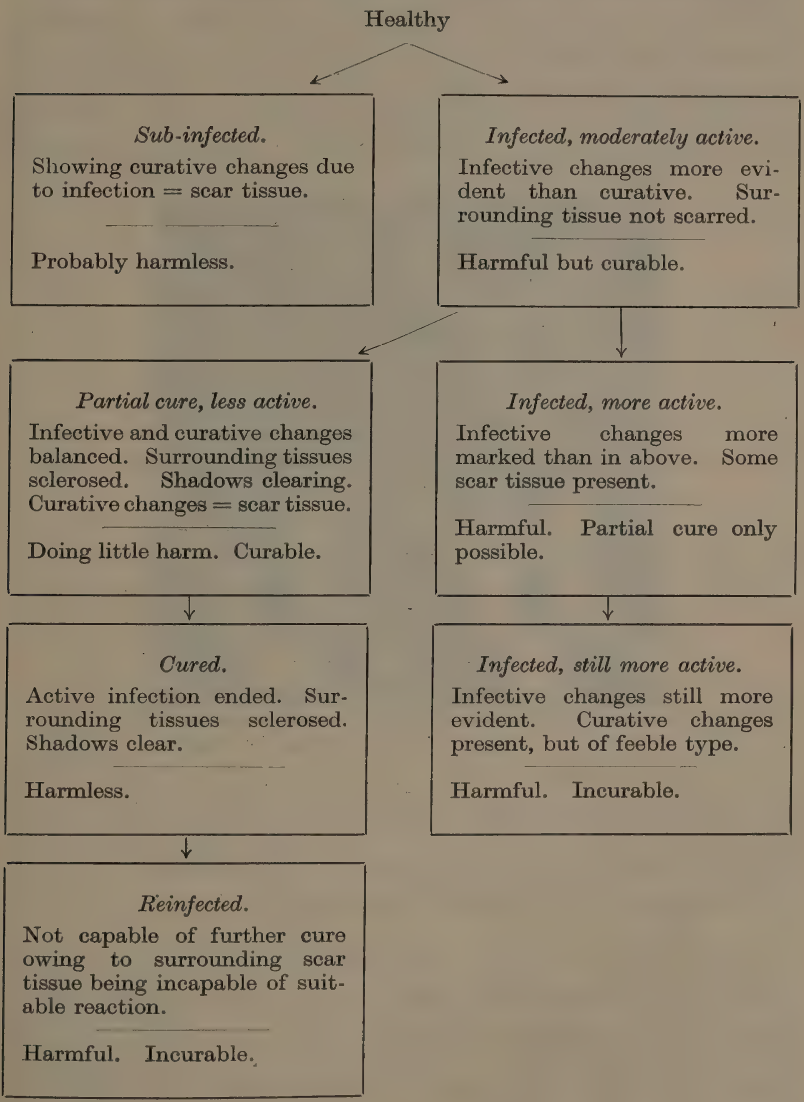
[1] *Brit. Dent. Journ.*, 1935, June 1 and 15, and 1936, Sept. 15.

If the infection is of an ephemeral character no appreciable changes can be discovered. But if it be of a more prolonged character, then the reaction of the tissues leads to changes in their shadows which to some extent it is possible to distinguish. In appearance these changes in the cancellous tissue vary considerably, but all are dependent upon the two inflammatory changes in bone known as rarefaction, or osteoporosis, and sclerosis, or osteosclerosis. According to which of these is the more active the changes seen will be either mainly destructive or mainly constructive. Each of these changes may be of a localised or of a more generalised character.

Before entering upon the description of the changes it will be a help to consider the accompanying scheme, which represents the type of lesions which may be present in relation to infected teeth and gums, and which it is desirable to be able to distinguish.

A Scheme representing approximately the Various Conditions which may be present in relation to Infected Teeth and Gums.



Healthy

Sub-infected.

Showing curative changes due to infection = scar tissue.

Probably harmless.

Infected, moderately active.

Infective changes more evident than curative. Surrounding tissue not scarred.

Harmful but curable.

Partial cure, less active.

Infective and curative changes balanced. Surrounding tissues sclerosed. Shadows clearing. Curative changes = scar tissue.

Doing little harm. Curable.

Infected, more active.

Infective changes more marked than in above. Some scar tissue present.

Harmful. Partial cure only possible.

Cured.

Active infection ended. Surrounding tissues sclerosed. Shadows clear.

Harmless.

Infected, still more active.

Infective changes still more evident. Curative changes present, but of feeble type.

Harmful. Incurable.

Reinfected.

Not capable of further cure owing to surrounding scar tissue being incapable of suitable reaction.

Harmful. Incurable.

The above scheme is not to be taken too literally, but merely to be regarded as approximately the types of lesions one may expect to find in dental films. From it it will be seen that the infection may be of a light character and do little harm, the reparative reactions keeping the infection localised—such an infection is called a sub-infection. Or from the first the infection may be moderately active and of a progressive character ; it is harmful, but still curable. Such lesions may undergo complete or partial cure, and after cure may become reinfected, or it may from the first become progressively more and more active until it is impossible to arrest its progress. It is important not only to remember these various stages, but to realise that they all may be, and generally are, present at the same time.

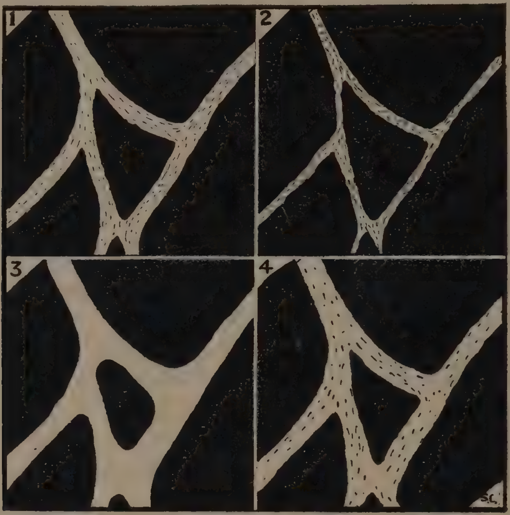


Fig. 585.—Gross changes in cancellous tissue.
1. Normal. 2. Porosed bone. 3. Sclerosed bone. 4. Hyperplasic bone.

Bone reacts to toxins, and certain other irritants, in two ways : it either becomes lighter or heavier, the former state being brought about by a thinning of the trabeculæ, the latter by a thickening (fig. 585). Thinning of the trabeculæ is generally accompanied by a thickening of the trabeculæ on the outskirts of the affected area, and is always followed by thickening as the irritant decreases in virulence or dies out. Thickening of trabeculæ may be the only change to the irritant. Thinning of the trabeculæ is known as osteoporosis, caries and rarefaction; thickening of the trabeculæ is known as osteo-sclerosis, or, more shortly, sclerosis. The former will show a decreased opacity to X-rays, the latter an increased.

Sclerosis.—It will be remembered that normal trabeculæ are not of uniform density, have a finely granulated structure, and that they meet each other at obtuse or acute angles, and thus a normal cancellous space is irregularly angular in outline. If the tissue undergoes a sclerosis the following changes occur: the trabeculæ thicken and become more uniformly dense, thus losing their granular appearance; the angles at which the trabeculæ meet become rounded, and thus the cancellous space becomes smaller and has a rounder outline. From a radiological point of view the structure of the bone is seen to have altered in appearance from an irregular angular mesh defined by delicate granular-looking trabeculæ to a roundish mesh defined by coarse uniform-looking trabeculæ—the character of the architecture has, if one may be allowed the illustration, changed from the Gothic style to the Norman. If this change has taken place in the absence of infection, the shadows will be bright and clean, although the bone will be more opaque; but if it has taken place in the presence of infection, then the sclerotic change will be imperfect, the trabeculæ being less dense and not fully formed, and in such cases the shadows will be indefinite and 'dirty.'

Porosis.—If the tissue undergoes porosis the trabeculæ become thinned and the cancellous spaces will increase in size, but their characteristic shape will remain. As the tissue is less dense, or rarefied, it will be less opaque to X-rays. Its shadow will be less dense, the trabeculæ will be more delicate and the cancellous spaces will be larger. At this stage the porosing process may stop and be followed by the sclerosing process described above. Or it may advance and the thinned trabeculæ at places are absorbed. In the jaws this absorption would appear to occur in multiple isolated spots each one of which will show as an area of increased translucency, or, more shortly, as an area of rarefaction. Some doubt has been cast upon the existence of these areas, but as it is difficult to explain certain changes in the bone in their absence their existence will be assumed. It is true that these delicate areas are difficult to see owing to the obscuring shadows of their enclosing bone, but diligent observers will be rewarded by finding them providing that the films they are examining are of good quality. As a rule the carious process does not advance beyond this stage and is followed by evidence of repair, but occasionally in more intense infections large areas of rarefaction appear. Such, however, are probably not due to the bacteria which ordinarily infect the jaw bone, and will not be considered as coming under the head of chronic infections.

Let us now consider more closely a tiny isolated area of rarefaction and follow its progress from formation to complete cure. The thinned walls of a few associated cancellous spaces are eroded through leaving behind a small irregular area of rarefaction, as shown in fig. 586. The process of repair now starts. Irregular bits of bone are absorbed and thus the irregularities in the outline of the space are removed. The wall of the space thickens, and gradually closes in upon it. This centripetal movement continues to diminish the size of the space until nothing remains but a small round dark spot surrounded by sclerosed bone. At the same time as these changes are occurring in the area itself reactive changes are taking place in the immediate surrounding bone, and these likewise consist of thickening of

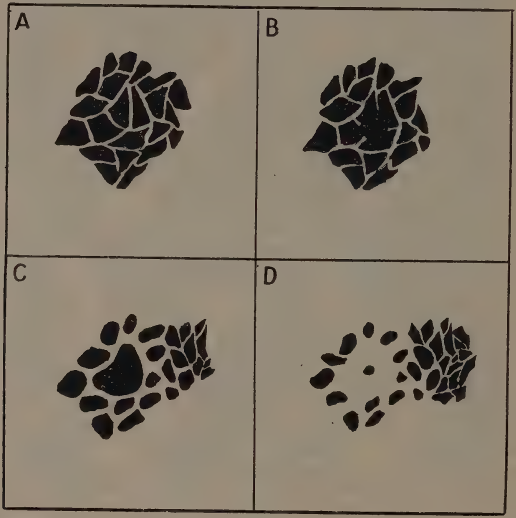


FIG. 586.—Ideal diagram of the formation and cure of a small rarefaction space. A. Normal cancellous tissue. B. Formation of small rarefaction space. C and D. Stages in its cure.

the trabeculæ and rounding of the cancellous spaces. Farther away still the pathological changes gradually cease until normal tissue is encountered.

The small round dark spots surrounded by sclerosed bone are diagnostic of old bone infection. Typical ‘ spots ’ are not always to be seen, even though present, unless the film, the rays, and the tissue are in suitable combination. They are not necessarily globular spaces, but may be oval or elongated, the roundish appearance being seen at certain angles only. They must be distinguished from nutrient canals. These are more ovoid, their surrounding bone not very thick and not uniform in density, and the trabeculæ which support them are not sclerosed. These inflammatory spots appear in some cases to undergo complete obliteration, leaving behind them nothing but a small mass of sclerosed bone—evidence of a cured mass of infected tissue. In yet other cases the process of cure occurs at an early stage, the area becoming surrounded by a thin layer of sclerosed bone and the space remaining more or less of the same size.

The radiographic evidence of the infective origin of these spots is indirect and is this : They are found in the neighbourhood of obviously infected roots, and they are frequently seen in the substance of an interdental septum which is in a state of chronic inflammation.

The *character* of the shadows is a matter of extreme importance in diagnosis. The aim of diagnosis should be not only to discover that an inflammatory lesion exists, but also whether it is active, quiescing, quiescent or sterile. Normal tissues give sharp clear shadows in which haziness is absent. Inflammation of bone due to infection gives no recognisable radiographic evidence of its presence in its early stages, but later there comes a slight haziness in the previously clear tissue shadows. This haziness is due to increased vascularity of the part and the presence of inflammatory products. The lesion being progressive, there follow destructive changes which show indefinite shadows which are hazy or dirty. The same type of indefiniteness accompanies early constructive changes, but as the infection dies down so do the shadows clear and the sclerosing tissue becomes more compact and definite. It is a fair deduction if the tissue shadows are clean and not dirty that the irritant effect of the infection has died down and that the lesion itself is sterile or quiescent.

Having now described in a general way the changes due to chronic infection that may be seen in the bones of the jaws, we will now discuss certain features of infection as it occurs either superficially from the surface of the mouth, or deeply from the apical foramina of teeth with infected or dead pulps.

(1) Infection from the Gingiva.—An infection from the superficial tissues may presumably at times remain localised, but in many cases it spreads deeply either directly through the surface of the bone or along the route of the immediate parodontal tissues.

(*a*) *Via the Surface of the Bone.*—An infection of the mucoperiosteal tissue involves the surface layers of the bone. If the infection is of a mild character only constructive changes are seen. Thus (fig. 587) the thin compact layer of the bone becomes thickened and sclerosed, and the superficial cancellous spaces show a diminution in size and a rounding of their irregular outline, changes due to thickening of their enclosing walls. Such a change may be limited to the superficial layers of the bone. Should the infection, however, be more intense, then destructive changes take the place of constructive. The surface layer of the bone is destroyed, and thus the superficial cancellous spaces are opened up. The normally clear shadows of the part may become a little hazy, but the tissues here are thin and unlikely to show this change. If the destructive changes persist, rarefaction of the deeper bone occurs and the area of destruction enlarges. But if the resistance of the tissues is sufficient the destructive changes give place to constructive. The free parts of the walls

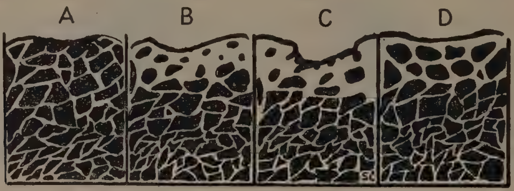


FIG. 587.—Surface infection of bone.

A. Destruction of compact surface layer, opening up of cancellous spaces.
B. Attacked bone has undergone repair. Sclerosed bone with roundish cancellous spaces.
C. The repaired bone has been re-infected.
D. A piece of bone in which infection and repair are comparatively balanced. The surface layer of bone is thickened, as are the trabeculæ, and the superficial cancellous spaces are rounded. Below is normal bone.

of damaged cancellous spaces become absorbed and the density of the remaining bone increases. A compact layer forms over the surface and the cancellous tissues below for a varying depth show rounding of their original irregular outline and their walls become thicker and more dense. Lower still the bone shows fewer sclerotic changes and gradually passes into normal tissue. The new sclerosed bone represents a healed tissue and may so remain, or it may later be reinfected, in which case the surface is again eroded and the cancellous spaces opened up, and a series of changes similar to those already described follow.

Infection from the surface is by no means always so limited, but, with or without destructive surface changes, may pass deep into the substance of the bone involving in certain cases an entire jaw. Depending upon the at present unknown characters of infections the changes seen will vary. Thus in some cases it would seem that numerous small colonies of bacteria settle in the tissues, forming in time small rarefaction areas which, undergoing the changes already described, leave behind a bone closely patched with sclerosed areas. In other cases the infection takes on a very extensive character and spreading widely may involve a whole jaw. They do not, in the experience of the writer, ever exhibit a generalised rarefaction of the bone, but always a generalised sclerosis, often of a very ‘ dirty ’ type, with here and there small patches of bone which would appear to be centres of infection. As a rule these cases are not the victims of a destructive marginal osteitis, but, on the contrary, may show a more or less unbroken though pathological surface bone. They are not very common, but they are most important cases, and can be easily missed on cursory examination (figs. 598 and 600 for type of sclerosis).

(*b*) *Via the Immediate Parodontal Tissues—the Periodontal Membrane and the Lamina Dura.*—From a gingivitis the infection and its products spread into the parodontal tissues, causing changes in both the bone and the periodontal membrane. The earliest stages of the infection cause no appreciable radiographic change. With persistence of the infection there comes a change in the periodontal membrane shadow ; it becomes hazy. Later the shadow becomes thickened. This thickening may be obvious or scarcely appreciable, and it may be general or localised to the cervical region, which is unusual, or to the apical region, which is usual.

At the same time as the changes in the periodontal membrane are occurring there are concomitant changes in the lamina dura. The increase in breadth of the periodontal membrane requires an enlarged housing space, and this is brought about either by a rise of the tooth in its socket, or more commonly by absorption of the dental wall of the lamina dura. If the infection be comparatively active the lamina dura will be eroded through at places and its continuity thus be broken. The breaking of the shadow is frequently seen about the apices of teeth, but it may be more general and in many cases it completely disappears. With a less active infection there will be a slight thickening of the periodontal membrane shadow, while the shadow of the lamina dura may show a decided thickening due to the filling up of the small cancellous spaces on its side away from the tooth. In still other cases the lamina dura, though unbroken, may show at irregular intervals erosions opposite which may be seen smaller erosions on the root shadow. They may be referred to as 'beading' of the periodontal membrane shadow (figs. 595 and 597). They are evidence of infection. They are usually found about the apices of teeth, but may be of a more general distribution. On the removal of a tooth with the affected membrane small erosions will be found on its root.

It follows from the above that a thickened periodontal membrane must be regarded as evidence of a progressing lesion, whilst a thickened lamina dura, of which the shadows are comparatively clear, is evidence of a quiescing lesion. Generally speaking, as the lesion quiesces the periodontal membrane will show a gradual narrowing, but its outline may be a little irregular, whilst the lamina dura will be irregularly thickened and will often merge into a patch of sclerosed bone (figs. 595 and 600). If the improvement continue the periodontal membrane undergoes further shrinkage, a change which is accompanied by an approach to the root shadow of a clear sclerosed surrounding bone which may have incorporated the lamina dura. At such a stage the lesion must be considered cured, or at least quiescent. But it is a damaged tissue, a fibrosed tissue, and should it again become infected there is little chance of a further recovery.

(2) Infection from a Dead or Dying Pulp.—Changes due to infection about the apices of teeth may be due to infection via the apical foramina or the periodontal membrane, and in certain cases it is impossible to distinguish the source. Thus it is not uncommon to see just beyond the apex of a tooth a faint rarefaction of a fusiform shape and lying in the direction of the main lymphatic drain. The rarefaction in these cases is due to a thinning of the trabeculæ. Such a change may be due to an infection from either route. It is evidently due to a low type of infection, or perhaps more correctly, as it is sometimes seen in relation to a tooth of which the pulp has died under a filling, to a low type of irritant. Should the infection cease, or decrease, the area becomes sclerosed.

In other cases in which the infection is more intense from the first, there is a thickening of the apical periodontal membrane. In time this membrane may show signs of beading and the lamina dura may disappear. The shadows will be somewhat hazy. Should the infection increase an acute or chronic abscess may form. A chronic abscess will show as a well-marked area of rarefaction surrounded by bone showing the usual changes due to infection. An acute abscess, with the exception of some blurring of the shadows, will have no radiographic signs. Should a patch of necrosis form, it will show no evidence in its early stages, but in later a dark irregular line will appear about the necrosed area and will indicate 'the line of demarcation.'

Granulomata.—Under certain conditions a granuloma may develop (fig. 594). A granuloma is a mass of soft tissue attached to the periodontal membrane. In a film it appears as an obvious translucency in relation to the apical portion of the root. It may or it may not be actively infected. If the infection has remained comparatively active the shadow of the surrounding bone for a varying distance will be rather dull and there may be some ill-defined thickening of the trabeculæ, but there will be no clearly defined limiting wall. As the intensity of the infection decreases the surrounding tissue shadows gradually clear, the more distant trabeculæ may narrow, while the closer remain thick and the bone immediately around the granuloma becomes condensed into a wall of varying thickness, the shadows of which in time become clearly defined. A granulomatous area showing these last characters is either sterile, or the infection has become satisfactorily encapsuled, and hence is practically harmless.

Granulomata may grow in relation to other parts of the periodontal membrane, more usually from the cervical region. They may be either on the lingual, buccal, anterior or posterior side of the tooth, or cover more than one side. If the granuloma is either on the lingual or buccal side it may be diagnosed with some degree of accuracy by the presence of a faint crescent-shaped line superimposed upon the shadow of the root, and having the convexity of the curve towards the apical part of the tooth. The shadow may extend across the interdental septum if the granuloma reaches in front or behind the tooth. These curved lines certainly suggest the presence of cervical granulomata, but alone they also represent loss of alveolar edge. The diagnosis is more certain if the surface of the bone of the related interdental septum shows blackish dots, which are characteristic of erosion spots. This important sign was first pointed out by Reginald Curnock (figs. 588 and 610).

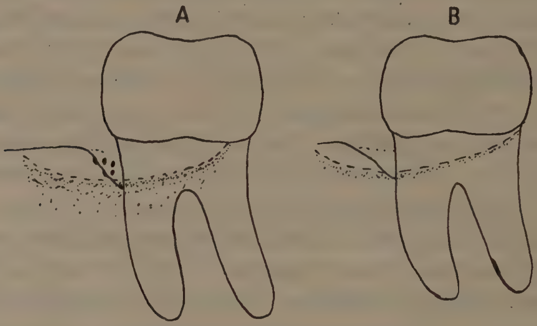


FIG. 588.—Cervical granuloma.

A. Note faint curved line of sclerosed bone with convexity towards apices of roots, and erosion spots on surface of bone of interdental septum. Suggests the presence of a granuloma on either the buccal or lingual and the anterior surfaces of the neck of the tooth.

B. Note same curved sclerosed line but without erosion spots. Might be a granuloma, but probably represents loss of bone only.

(See also Fig. 604.)

A granuloma may become cystic. If it is sterile it will show characters similar to a sterile granuloma, except perhaps that the rarefied area will be more translucent. It will differ from an ordinary uninfected cyst in that the wall of the latter will be narrower and of more delicate structure and it will be supported by a cancellous tissue of normal structure. Further, if in each case the periodontal membrane shadow be followed round the root, it will be found that the shadow of the granuloma or cyst which has arisen from it will blend with it, whereas in the case of a cyst unconnected with the root it will remain separate. If an ordinary cyst becomes infected its wall in time will show infective changes.

(3) Changes of Uncertain Origin.—(*a*) *Osteoid Sclerosis* (fig. 589, see plate A facing p. 580).—The name osteoid sclerosis is used for want of a better. In this variety the characteristic cancellous pattern of the bone is replaced by a tissue the shadow of which gives the appearance of finely ground glass. The laminal dural shadows have disappeared, and as a rule the periodontal membrane shadows show a little thickening. The intersocket bone may show a less dense streak about midway between the sockets. The complete obliteration of empty sockets may be delayed. It is not a true sclerosis of bone in the ordinary sense of the word, but represents an osteoid condition possibly brought about by a special infection. (The condition requires investigation.) It is frequently seen in interdental septa which have been infected over a long period, and especially in patients who by following a careful hygiene have kept the disease more or less in abeyance. It may occupy a whole jaw, usually the mandible. It is mentioned here rather than under inflammatory conditions so that its appearances can be contrasted with diffuse osteitis deformans, from which it must be distinguished.

(*b*) *Diffuse Osteitis Deformans.*—In the last edition this disease was described as diffuse osteitis fibrosa, but as it may be found with other manifestations of Paget's disease, it seems more correct to describe it under the above title. The disease usually appears in the maxilla. It differs in appearance from the diffuse osteoid sclerosis in that its ground glass appearance is much coarser, and the whole mass of affected bone is much more uniform in appearance, and here and there—and this is a diagnostic point—will be seen flaky, more opaque areas. After the extraction of a tooth the empty socket is rapidly filled with the pathological tissue and all trace of the empty socket disappears. The periodontal membranes are not as a rule thickened.

(*c*) *General Sclerosis* (*Hyperplasia*).—This variety, the independent existence of which is perhaps uncertain, occurs in vigorous types of patient, such as used to be described as ‘ gouty ’ types, and it is probably part of associated changes in other parts of the body. The characteristic points about it are: the shadows are clear; the trabeculæ are thickened but do not present a sclerosed appearance; the angles at which they meet remain normal and the cancellous spaces retain their usual shapes. Its cause would appear to be due to a mild irritation which stimulates growth, and the origin of which is outside the mouth.

(*d*) *Other Conditions.*—It is not proposed to make an attempt to describe other conditions seen on dental films, and which do not conform to descriptions already given. It is necessary, however, to warn the student that they do occur and that the origin of them is uncertain. When he does discover them, they will offer him an opportunity of exercising his scientific acumen.

(D) THE RELATION OF LYMPHATIC FLOW TO THE EXTENSION OF BACTERIA AND THEIR PRODUCTS.

It is accepted that the direct extension of bacteria and their products is bound up with the direction of the lymphatic flow. The study of dental films suggests that the spread of infection from localised lesions follows a more or less main drainage which has a very free anastomosis with neighbouring parts. This radiographic evidence is strongly supported by the recent and very interesting work of Alexander MacGregor.[1]

The laboratory evidence has shown that the lymphatics from the gingival trough pass down the periodontal membrane, anastomosing freely *en route* with those from adjacent parts, and at the apex of the tooth anastomose with those from the pulp. And further, that the lymphatics from the gingiva pass through the bone to the pulp, dentine and cementum of adjacent teeth. The net result of the evidence is that the lymphatics of the bone, teeth and mucous membrane anastomose freely.

The evidence of radiology as to the flow of the lymphatics is instructive and has clear clinical bearings (figs. 590 (see plate B facing plate I) and 610). If it be allowed—and it must be allowed by the most sceptical—that the main infection follows the course of the main lymphatic drain, and leaves behind in its wake a permanent record of its distribution in the shape of altered tissue, the following deductions can be drawn: The lymphatics from the maxillary incisors pass to below the floor of the nose—a direction also supported by the occasional occurrence of an abscess in that region in relation to infected incisors. Those from the canine pass upwards and backwards towards the floor and anterior wall of the antrum, which sinus it occasionally infects. Those from the premolars and molars pass upwards—the sinus being frequently infected from the buccal and occasionally from the palatine roots of the molars. In the mandible the lymphatic flow from the mandibular incisors passes downwards and ends in the submental gland, whilst those from the remaining teeth pass downwards into the mandibular canal.

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[1] *Proc. Roy. Soc. Med.*, 1936, xxix, 1237

The study of the films suggests also that the teeth might be arranged in certain groups. These observations have a clinical bearing which is of more importance in the case of the maxilla than the mandible. Thus, if a maxillary first incisor is infected it is comparatively certain that the infection will spread to the second incisor of the same side and vice versa. If the canines and premolars be infected, then the teeth in front and behind this group may escape; for example, an infected maxillary second premolar may not infect a first molar. Further, if M. 1 be infected M. 2 will almost certainly suffer, whilst the septum between M. 1 and P.M. i may escape (fig. 608). If M. 2 be infected M. 1 may escape, but M. 3 is likely to be infected. The evidence of this spread of infection from tooth to tooth is very strong, and certainly suggests that infected teeth showing indifferent signs of recovery should be removed if adjacent teeth are to be saved. In children infection spreads more rapidly than in adults, so that retention of infected teeth in them cannot be justified.

In connection with the lymphatic flow it seems appropriate to discuss here the so-called ‘lymphatic streaks’ which may often be seen in the bone between teeth. These streaks of less opaque bone pass inwards between the roots of teeth in the substance of the interdental septa. They are commonly seen between the mandibular incisors, but may be seen elsewhere (fig. 609). The probable explanation of these markings is ‘that they are the remains of the interfollicular bone, and that they are seen because the junction between the adjacent tooth follicles, each of which has its own lymphatic drain, is imperfect’ (S. Colyer). Further. if they were in the position of the lymphatic drain of the part they would suffer changes due to infection such as are seen so frequently in the bone beyond the ends of infected teeth. Far from so reacting, they often retain their normal bony structure even though in relation to bone showing evidence of infection. It is probably correct to regard them as a congenital defect due to imperfect fusion of adjacent tooth follicles.

(E) RESIDUAL INFECTION.

Resolution of infected jaws may be complete or incomplete. The time of resolution cannot be foretold from radiograms, but, speaking generally, they suggest that most residual infections undergo satisfactory resolution within two years. In complete resolution no evidence of the previous infection remains; but in incomplete resolution the infection remains more or less generalised or limited to certain areas. Resolution may be incomplete in the sense that structural changes in the bone brought by the previous infection to some extent remain, but it does not follow that the infection itself may not be cured, or quiescent. Such incomplete resolution is common in jaws which have suffered a marked sclerosis, and clinically corresponds to edentulous jaws showing bossy irregular surfaces. But resolution may be far from complete, the whole jaw showing evidence of comparatively active infection. The unfortunate condition known to dental surgeons as 'persistent shrinking of the jaws' is almost certainly due to residual infection, and would appear to continue until the infection comes to an end.

It will be seen from the above that it must not be assumed that because the edentulous bone does not return to normal that the jaw is still infected. All chronically infected bone has undergone some degree of fibrosis and sclerosis, and in such cases the end results of repair will be abnormal tissue and consequently give abnormal radiographic shadows.

Of the causes which give rise to unresolved infection the commonest is, perhaps, the insufficient extraction of infected teeth, which, if in relation to an area of infected tissue, continually reinfect it. Other causes are: incomplete extraction of infected roots; infected cyst walls; and lastly, lack of resistance on the part of the patient.

Apart from the remarks that have already been made, residual infection can be diagnosed comparatively safely if the usual evidence of active chronic osteitis be present. Other evidence is the failure of the edge to form a compact surface layer—this is the ‘ saw edge ’ of certain writers. There are, however, many cases in which only a bacteriological or histological examination can settle a doubt.

Lastly, in connection with residual infection it may be said that radiology gives little or no sanction to the surgical treatment of chronically infected jaws. Once the main causes are removed resolution of a satisfactory character in healthy patients is practically certain ; in unhealthy it is difficult to see how it would be aided by such interference.

(F) THE RELATION OF RADIOGRAPHIC FINDINGS OF INFECTION TO CLINICAL SIGNS.

Radiographic changes in the bone do in some cases bear a relation to clinical signs to be observed in the mouth, but at present this side of the subject has not been properly worked out and much remains to be done. On this difficult subject the following observations may be made with some degree of assurance.

No changes in the bone are to be observed in the early stages of gingivitis, however severe it may be. Later, the edge of the alveolar process is destroyed, and the periodontal membrane shows some thickening, which may be more or less general or may be limited to the apical or cervical region. Still later, although there is no apparent loss, or shrinkage, of the interdental papillæ, the alveolar edge may show well-marked destruction with the formation of potential pockets ; that is to say, the bone in relation to the necks of the teeth shows destruction, or loss of lime salts, in the position where a clinical pocket will ultimately be found. These are important points because they show that bone destruction precedes gum recession, and so when clinically there is found shrinkage of the apices of the interdental papillæ the clinical sign must not be considered as indicating an early stage of the disease but a comparatively late one.

With the improvement of the superficial lesion there will be found reparative changes in the deeper tissues. The edge of the bone and the walls of the pockets undergo sclerosis. This sclerosis may affect not only the immediate parodontal tissues but may extend deeply into the bone, emphasising in so doing the extent of the original infection.

A well-marked sclerosis of bone following upon a cured, or partly cured, parodontal disease corresponds to a fibrotic condition of the gum margin, which fibrosis may be limited to the tips of the interdental papillæ or may extend over their whole surface. A fibrotic gum margin therefore suggests a sclerotic bone edge, and is, to some extent, evidence of a chronic lesion which is becoming, or has become, quiescent.

As the superficial lesion is the primary source of the infection of the deeper tissues, the healing of the latter naturally lags on that of the former, and though it generally in time shows signs of repair, there are a few cases in which it seems unable to recover. It is indeed not uncommon to find an apparently healthy mouth with well-marked signs of infection of the deeper bone. If such a change be found radiographically, it is as well to re-examine the case clinically to ascertain if there are any signs of slight looseness of the teeth, or whether they are dull on percussion. Such a case may, or may not, ultimately recover: In such cases experience is the only guide.

In all toxæmic cases, especially in young persons, a radiographic examination of the jaws is called for, especially if the mucous membrane is somewhat bluish, a little swollen, a little sticky and, perhaps, shows dilated venules at places. It is not uncommon in such cases to find on radiographic examination a comparatively severe general infection of the bone. A cursory examination of the films may show but little change, and they are often passed as normal. But a more careful examination will reveal that the detail of the tissues is a little hazy, that of the laminæ duræ about the roots have disappeared, that some of the periodontal membranes are a little thickened and here and there may show signs of 'beading.' The diagnosis is important because there seems no doubt that many such cases if placed under suitable conditions do undergo cure to a large extent. The difficulty for the dentist is to know when the stage of possible cure is passed.

It will be clear from the above that the degree of jaw infection bears no very obvious relation to clinical signs. Thus, on the one hand, the alveolar edge may be practically intact, local clinical evidence may be so fine as to elude the observer, and yet there may be severe infection of the deeper tissues. On the other hand, alveolar edge destruction may be marked, local marginal gingivitis severe, and yet the deeper tissues may show little or no radiographic evidence suggesting infection. It is instructive to note that the first type usually occurs in youngish persons, who will often be found to be suffering from some degree of toxæmia, whilst the second type occurs in older persons, those beyond middle life, and as a rule their general condition is fairly satisfactory. The important deduction to be drawn from these observations is that chronic infections of the jaws, the ill-effects of which are so often seen in early and late adult life, is often a disease of early life, the inception of which may date back to any period following the eruption of the teeth. Radiology indeed suggests that infection occurs rapidly in youth and that the older we get the more resistant do our tissues become.

(G) CERTAIN CHANGES IN THE ROOTS OF TEETH.

(1) Proliferative or Productive Periodontitis (Exostosis).—Exostosis, or productive periodontitis, is seldom limited to one tooth. As a rule symmetrical teeth are affected, and in some cases every tooth present may show signs of the condition. The accepted pathology is that it is a productive periodontitis due to infection from the periodontal membrane. Radiology does not support this contention. The evidence of radiology is that it is not usual to have evidence of an infection unless the tissues around show signs of reaction. In cases of productive periodontitis, therefore, one would expect to find evidence of infection in the adjacent interdental septa and periodontal membrane. Though in the majority of cases this is so, in a minority the septum appears to be normal, and moreover it is not uncommon to find an unerupted exostosed tooth surrounded by normal bone. It must be pointed out that the presence of a related infected interdental septum is not evidence of its relation to the exostosis as it is not a universal relation[1] (figs. 605, 606 and 607).

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[1] There may be two forms of exostoses, one of which, the more common, has no relation to an infected periodontal membrane. This form is probably always symmetrical, frequently multiple, and is associated with normal-looking periodontal membranes and normal interdental bone. It may occur in unerupted teeth

With regard to the clinical importance of exostoses in reference to infection the following remarks may be made. As radiology does not support the contention that most cases are due to infection, it does not suggest that they are doing harm to the patient. The following suggestions may be taken as a guide to treatment (fig. 591) :—

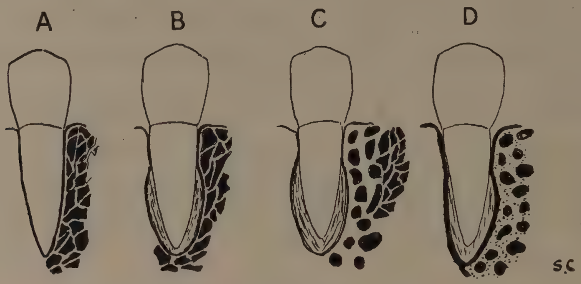

FIG. 591.

A = Normal.
B = No evidence of bone infection.
C = Periodontal membrane shadow not thickened, adjacent bone sclerosed but its shadows clear.
D = Thickened periodontal shadows, adjacent sclerosed bone shadows are hazy.

(*a*) If the periodontal membrane shows no thickening, and the associated bone no evidence of infection, the tooth cannot be regarded as harmful. Fig. 591 B.

(*b*) If the periodontal membrane shows thickening associated with evidence of rather recent infection of the adjacent bone, the tooth is infected but may recover.

(*c*) If the periodontal membrane is normal, or even thinner than normal, and the adjacent bone shows some sclerotic changes, the shadows of which are clean and sharp, then the infection is either cured or quiescent, and is probably harmless. Fig. 591 C.

(*d*) If the periodontal membrane is thickened, and the adjacent bone shows sclerotic changes, the shadows of which are dirty and indistinct, it is evidence of a reinfection of a cured or partially cured tissue, and the condition is one almost certainly incurable. Fig. 591 D.

in uninfected bone (fig. 607). Further, this form is often associated with pulp stones which are remarkably common and generally symmetrical. Such forms of exostoses may be hyperplasias, and the cause leading to them may be outside the mouth (S. Colyer).

(2) Rarefying Periodontitis (Absorption).—Absorption of the apex of a tooth may occur in a previously exostosed tooth or in one that was previously normal in appearance. In each case the diagnosis will rest upon the abnormal outline of the root. In some cases the periodontal membrane shadow shows thickening and irregularity, and the adjacent bone shows infective changes. Such teeth are actively infected. But there are others in which the periodontal membrane shadow is not thickened or irregular, and in which the bone adjacent to the root does not appear to be infected.

(3) Translucency of Roots.—Translucency of roots cannot be diagnosed by radiography.

(4) Bony Attachments of Teeth to the Jaws (Ankylosis).—This condition is rare. It is diagnosed by noting that the shadows of the root and the surrounding structures merge.

(H) INFECTION OF THE MAXILLARY SINUSES.

Infection of a maxillary sinus is occasionally of dental origin, the infection coming, as a rule, from a buccal and sometimes the palatine root of a first or second molar, and occasionally from the root of a canine. It is said to arise at times from the roots of the premolars. The evidence of radiology is that the infection may arise from an apical abscess, which opens directly into the floor of the sinus, or that it may arise in a less obvious way by the spread of a low type of infection through the bone between the apices of the roots and the floor of the antrum, and there bring about a chronic infection of its muco-periosteum.

A diagnosis of infection of an antrum may occasionally be made by means of intra-oral films. A loss of translucency is in favour of infection. Care in coming to a conclusion is necessary as the outer wall of an antrum frequently shows increased density due to chronic infection from teeth and gingiva, and such increased density may be unilateral. The discovery of an abscess at the apex of a tooth associated with a decreased translucency of the antrum is in favour of a positive diagnosis. Other causes of increased opacity of an antral shadow are antral polypi and, rarely, malignant growths. The diagnosis is usually made by means of extra-oral films (fig. 592, see plate B facing plate I). By this means the two antral shadows being on the same film a comparison can be more fairly made. A moderate increase in the opacity of an antrum suggests an infected sinus but not necessarily pus. A more dense opacity is in favour of pus, but might also indicate polypi or other growth. Some opacity and a more opaque shadow with a horizontal line above the floor of the antrum is the only certain diagnostic evidence of fluid in the sinus. Any doubt can be set aside by re-radiographing the patient with his head in the same relation to the casette (as given before) but with vertical axis turned to one or other side. If there is a 'fluid level' it will be seen as a horizontal line, and therefore will be, in this instance, oblique to the floor of the antrum.[1]

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[1] With reference to the spread of infection from the apex of a tooth to the floor of the antrum it should have been mentioned in the text that the probable effects of the chronic infection of the umco-periosteum of the floor of the antrum are immobilization and destruction of the ciliated epithelium over the affected area leading to stagnation of the antral secretion.

PLATE A.

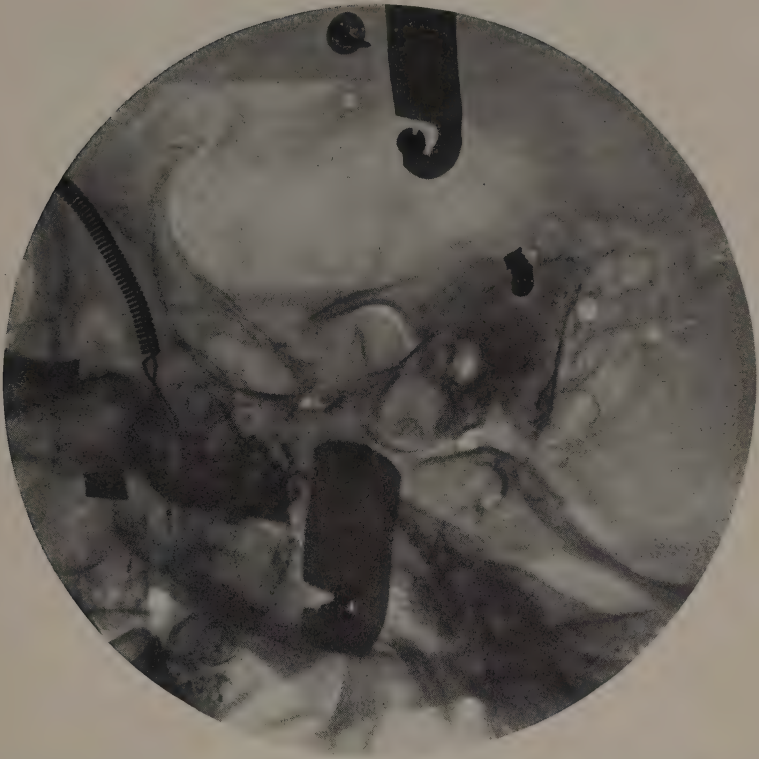

FIG. 582.—Temporo-mandibular articulation—from a dried skull. The articulation is seen just in front of the external auditory meatus, and behind this are seen the mastoid air cells. In front of the articulation is the zygomatic process. The articulation is seen from above at an angle of 15. (Charles Colyer.)

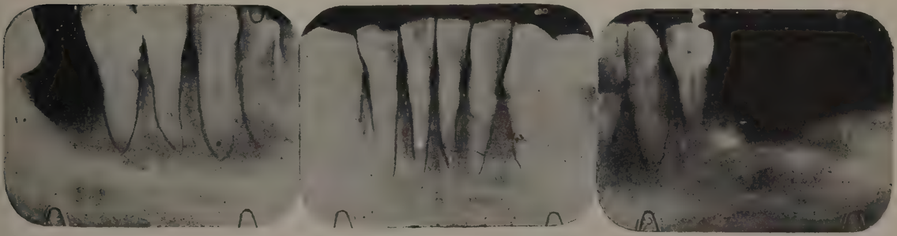

Fig. 589.—Osteoid sclerosis. Note the diffuse type of sclerosis which has entirely replaced the pattern of the normal cancellous tissue ; the absence of laminal dural shadows ; the less dense streaks of bone in the interdental septa ; and the slight thickening of the periodontal membrane shadows.

PLATE B.

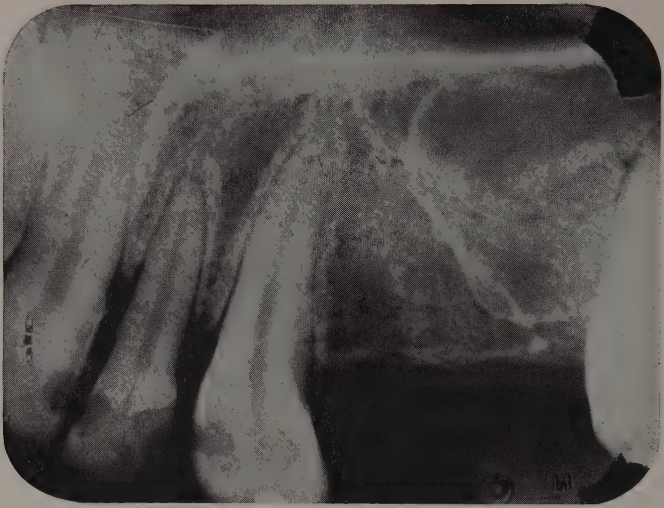

FIG. 590.—Bone changes in the direction of the lymphatic flow. Note the rather dirty sclerotic bone above the aspices of $|\underline{23}$.

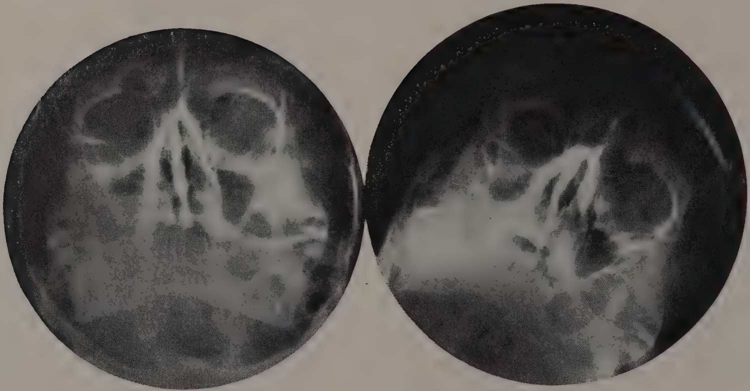

FIG. 592.—Infected antrum showing fluid level.

Left figure. Shows head in vertical position. Horizontal fluid level in left antrum.

Right figure. Head oblique. Note that top of the dark shadow in left antrum is still horizontal.

(Charles Colyer.)

PLATE I.—RADIOGRAMS.

Examine these prints in a moderately strong reflected light.

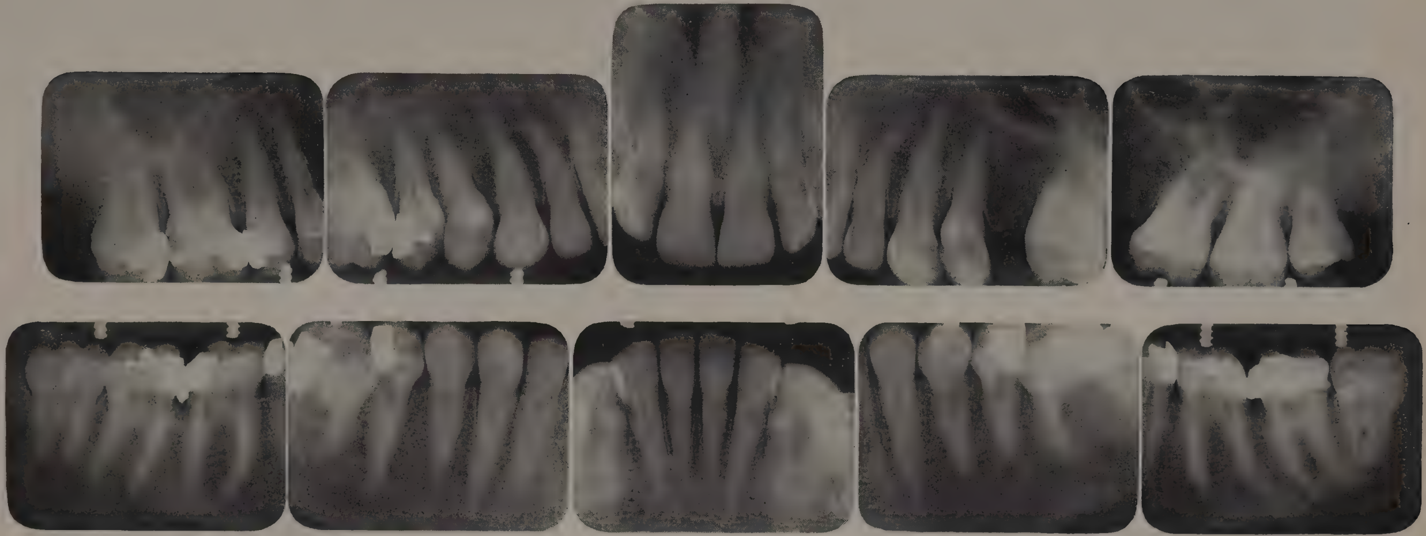

FIG. 593.—Comparatively normal. The tissues about the apices of $3|3$ and $\overline{1|1}$ are infected, and those about the maxillary incisors show reactive changes due partly to infection and partly to injury due to the regulation of these teeth about the age of twelve years. All normal appearances can be seen in these films. Patient aged about twenty-five years.

PLATE II.—RADIOGRAMS.

Examine these prints in a moderately strong reflected light.

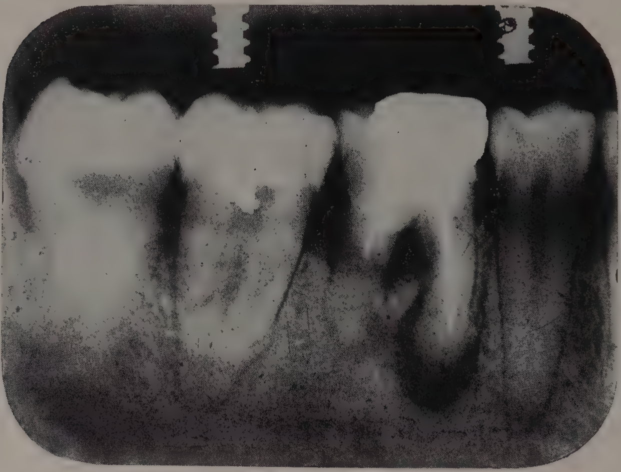

FIG. 594.—Granuloma at the apex of the anterior root of $\overline{6|}$, and absorption of the posterior root. Thickened irregular periodontal membrane shadows about $\overline{5|}$ and $\overline{7|}$. Obliteration of the laminal dural shadows and sclerosis of the parodontal bone.

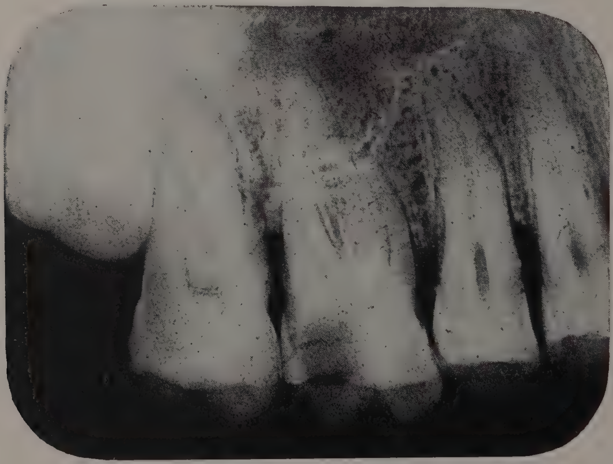

FIG. 595.—An example of ‘ beading ’ of the periodontal membrane shadow, especially about the root of $\underline{5|}$. The parodontal tissues show evidence of an old infection, as suggested by the marked sclerosis. Note also the sclerosis of the wall of the antrum.

PLATE III.—RADIOGRAMS.

Examine these prints in a moderately strong reflected light.

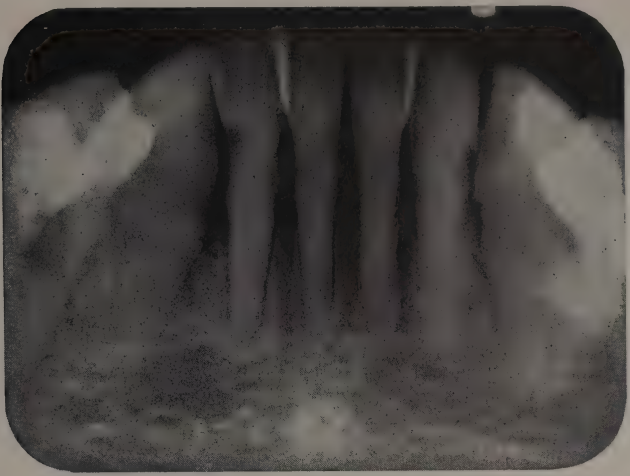

Fig. 596.—Note the thickened periodontal membrane shadows about the mandibular incisors; the thickening of the laminal dural shadows; the patch of sclerosed bone round the root of $\overline{2|}$. These are chronic infective lesions of a comparatively active character.

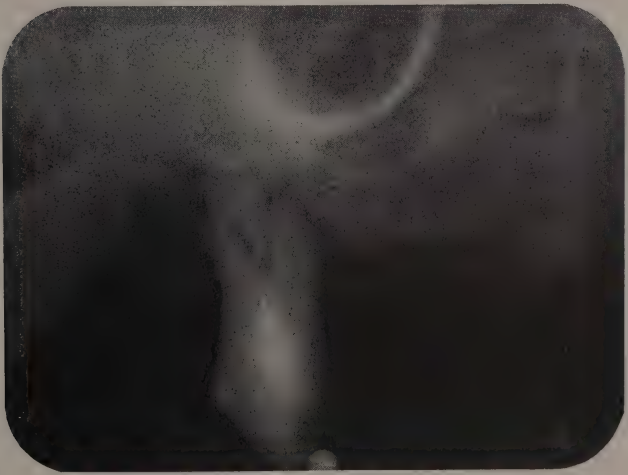

Fig. 597.—Shows in the original film haziness of the bone about the roots of $7|$. The periodontal membrane shadow is thickened. It is an active lesion. Note also small irregular areas of rarefaction surrounded by sclerosed bone.

PLATE IV.—RADIOGRAMS.

Examine these prints in a moderately strong reflected light.

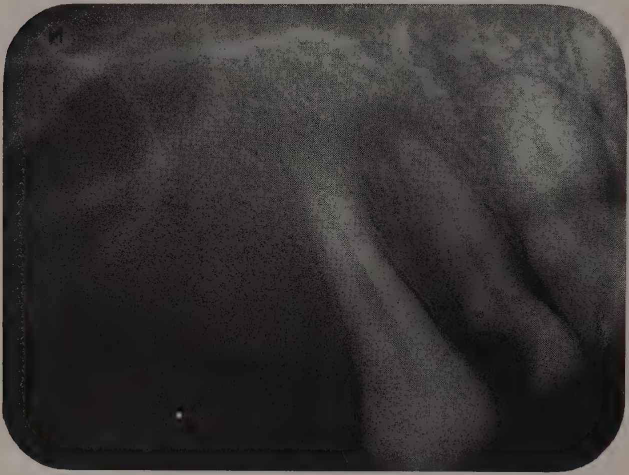

FIG. 598.—Note especially the indefinite rarefaction of the bone at the apex of $\underline{2|}$. There is no suggestion of the ‘ walling off ’ of this lesion. Active.

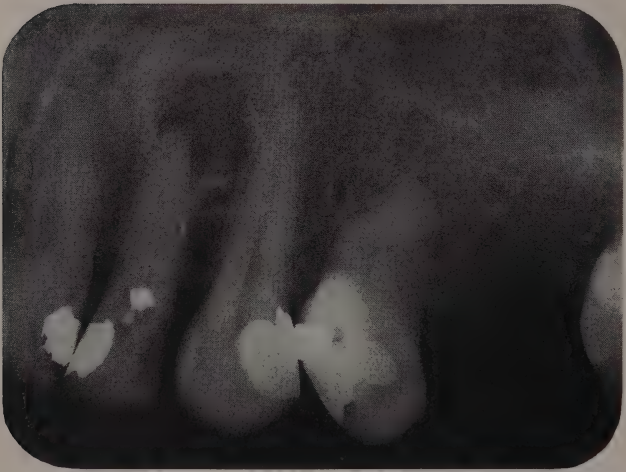

FIG. 599.—Note especially the rarefaction of the bone at the apex of |2. There is a decided sclerosing of the bone about it, suggesting a lessening activity of the lesion.

PLATE V.—RADIOGRAMS.

Examine these prints in a moderately strong reflected light.

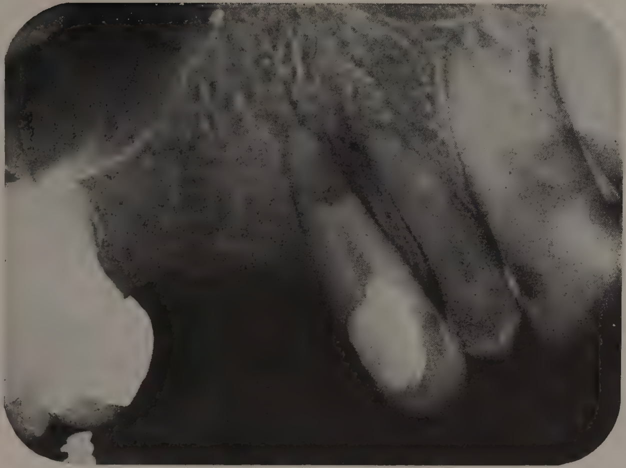

Fig. 600.

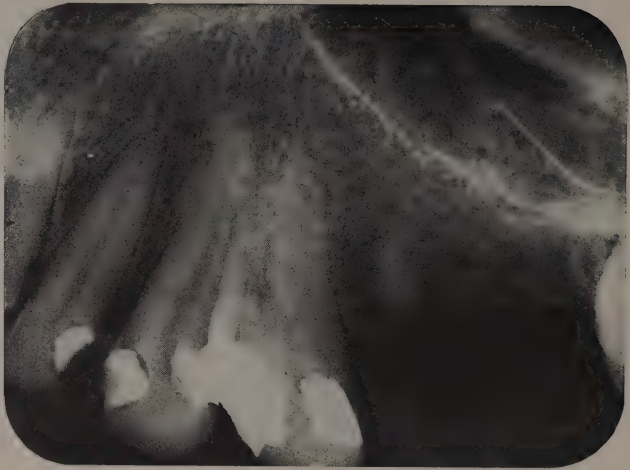

Fig. 601.

Figs. 600 and 601.—Two films from the same patient. The tissues on the right side show a more advanced condition due to infection than those on the left. Note the thickened trabeculæ and the rounding of the cancellous spaces. Ill-defined small rarefaction areas can be seen on the right side. Lamina dura of 1| merging into sclerosed bone of interdental septum.

PLATE VI.—RADIOGRAMS.

Examine these prints in a moderately strong reflected light.

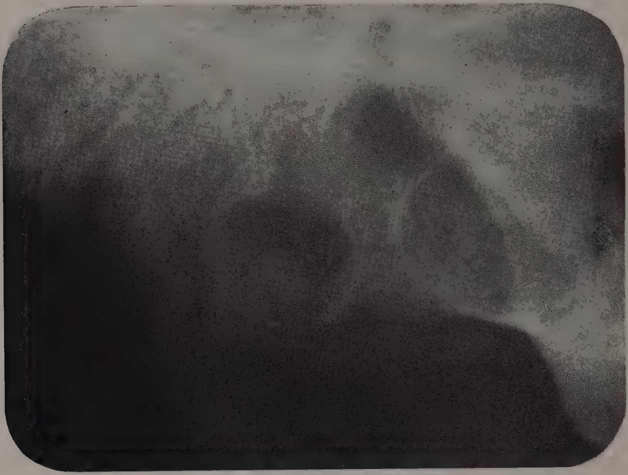

Fig. 602.—Shows a circular translucency surrounded by a very thin line of bone. It is almost certainly an uninfected cyst. The bone in front of this cyst shows changes due to infection ; it is probably still infected.

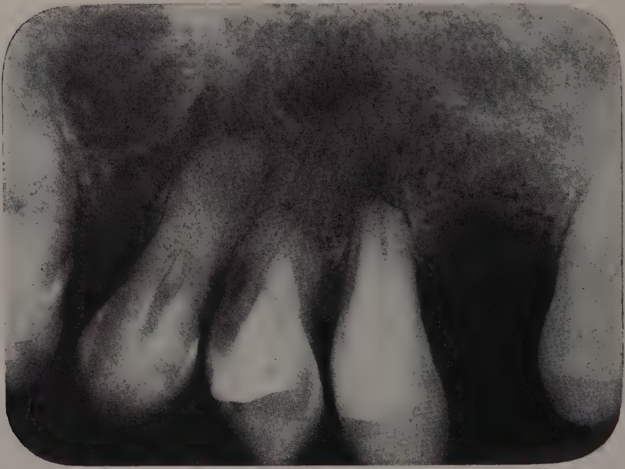

Fig. 603.—Note the large irregular mass of rarefied tissue above ⌊3 and ⌊4. Most of the bone is sclerosed. In the bone in front of ⌊3 can be seen small irregular areas of rarefaction which are in varying stages of cure. (The earlier stages of small rarefaction areas cannot be reproduced satisfactorily.) Observe the rounding of the cancellous spaces, and that the infected bone reaches to the wall and floor of the antrum.

PLATE VII.—RADIOGRAMS.

Examine these prints in a moderately strong reflected light.

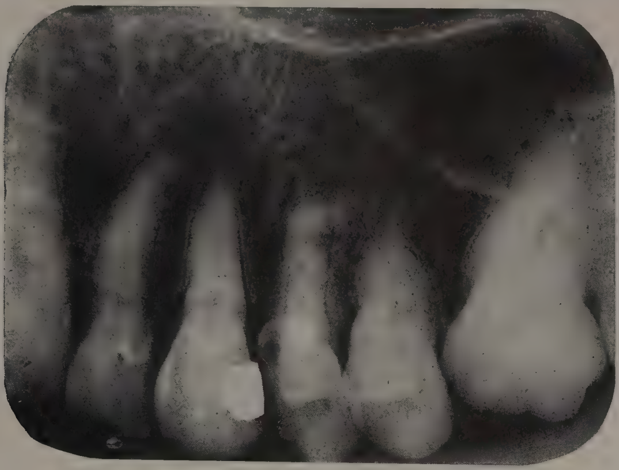

Fig. 604.—Note the faint area of rarefied tissue above the roots of |2 and |3. The whole of the tissue above |2345 is abnormal. Observe the rounding of many of the cancellous spaces, ' black spots,' and the irregular thickening, where not absent, of the lamina dura towards the apex of |3.

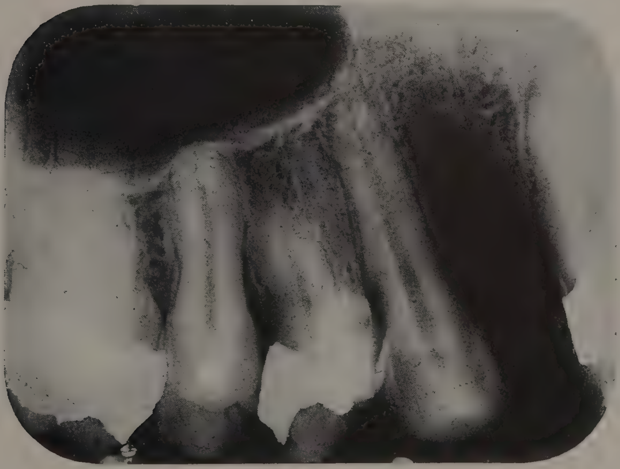

Fig. 605.—Note small, round, dark spots surrounded by sclerosed bone in the septum between 6| and 5|, and opposite the exostosis of 5|. Whether this exostosis was due to inflammation of the septum, or whether the septum has been subsequently infected, is uncertain. The sclerotic character of the bone should be noted. From a ' gouty type ' of patient.

PLATE VIII.—RADIOGRAMS.

Examine these prints in a moderately strong reflected light.

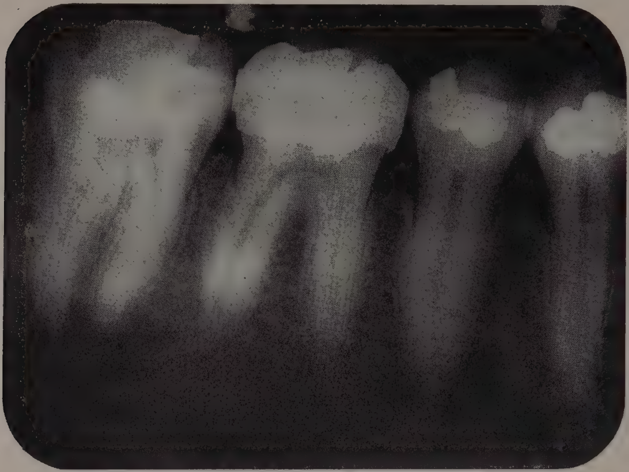

Fig. 606.—Note exostosis of the root of $\overline{5}|$. There is some loss of bone about the neck of $\overline{5}|$ indicating some infection, but there is no thickening of its lamina dura, or evidence of infection of the adjacent bone.

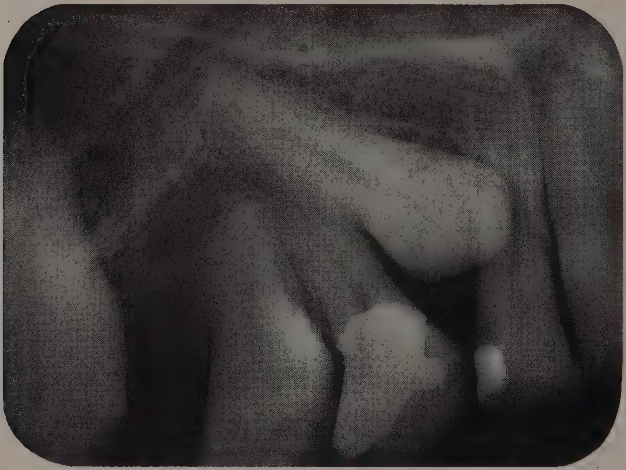

Fig. 607.—Shows an unerupted exostosed $\underline{3}|$ in a patient aged about thirty-five years. There is no evidence of infection of the bone about this root, although there is some infection about the roots of $\underline{5}|$ and $\underline{4}|$. The patient was of the 'gouty type' and very subject to fibrositis, otherwise he was a healthy type.

PLATE IX.—RADIOGRAMS.

Examine these prints in a moderately strong reflected light.

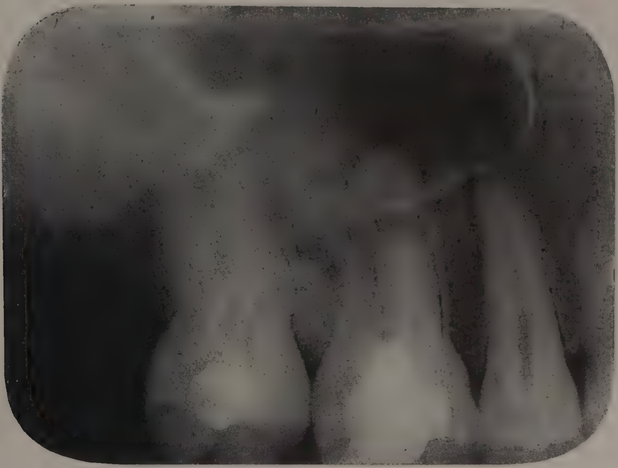

Fig. 608.—Note rarefaction areas over the apices of palatal and buccal roots of $\underline{6|}$. Also that the septum between $\underline{6|}$ and $\underline{5|}$ has partly escaped infection, whilst that between 7| and $\underline{6|}$ is infected.

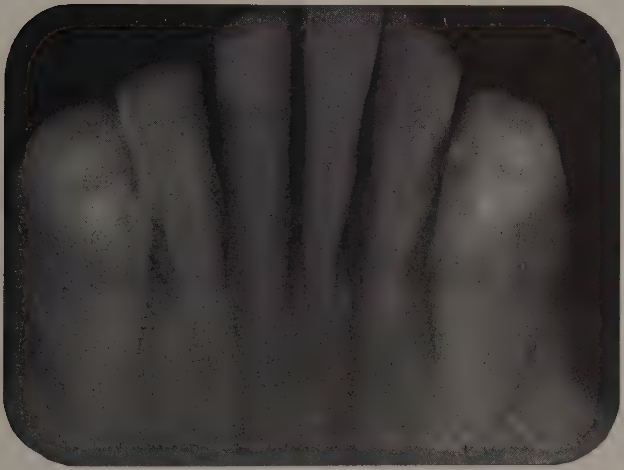

Fig. 609.—So-called ‘Lymphatic Streaks.’ For probable explanation see pages 573–574.

PLATE X.—RADIOGRAMS.

Examine these prints in a moderately strong reflected light.

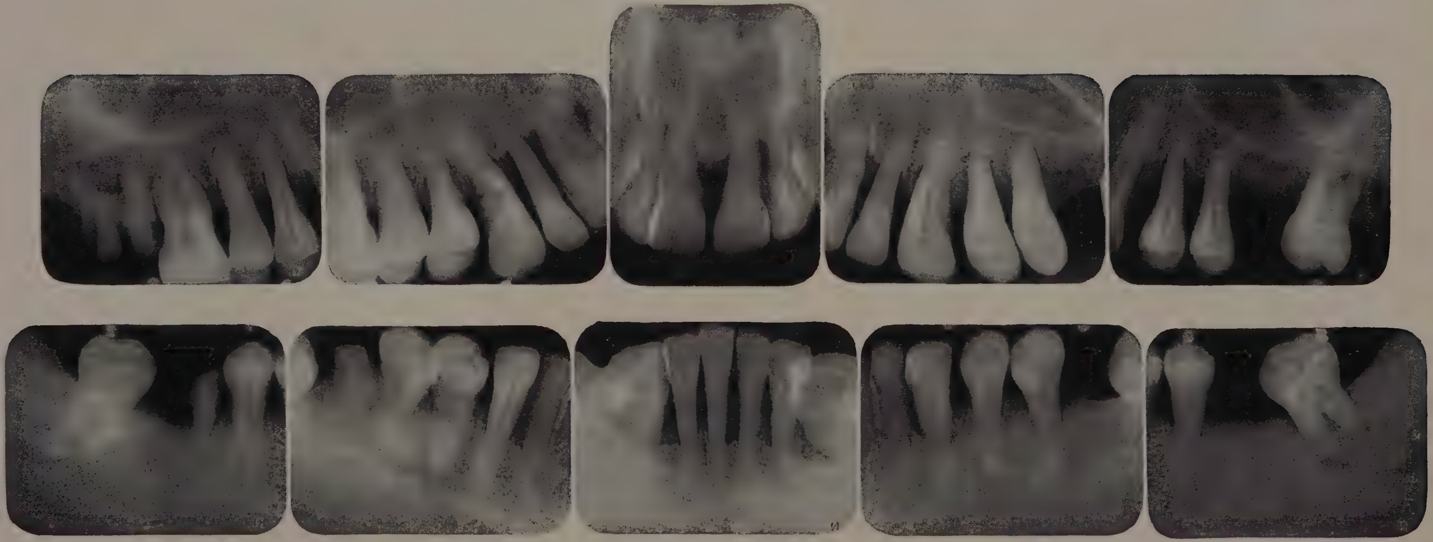

FIG. 610.—A case of advanced parodontal disease.

Note : Considerable destruction of the alveolar edges ; the thickened periodontal membrane shadows ; the alteration in pattern of practically all the cancellous tissue ; and the lost, thickened, or broken laminal dural shadows. The films suggest a long-standing case of parodontal disease which, to some extent, has been kept in abeyance by careful treatment, but which is slowly progressing. Some of the curved shadows on the roots just below the present alveolar borders may represent cervical granulomata.

CHAPTER XVIII

Parodontal Disease. Synon.: Pyorrhœa Alveolaris.

The Anatomy of the Parodontal Tissues—Clinical Appearances—Morbid Anatomy—Pathology—The Disease in Animals—Bacteriology—Etiology—Treatment—Sequelæ.

THIS disease is a chronic infection of the gingiva and adjacent tissues; it is characterised by a progressive destruction of the alveolar bone and the periodontal membranes, which destruction is accompanied by a recession of the gum margin, and in the final stages of the disease by extrusion of the teeth. It is accompanied sometimes in the earlier and almost always in the later stages by a free discharge of pus around the teeth involved.

It has been taught for many years by one of us (J. F. C.) that this disease commences as a marginal gingivitis; that it is so is confirmed by the histological work of James and Counsell, which is in turn confirmatory in many respects of the work of Gottlieb and Fleischmann; it does not start primarily in the periodontal membrane, and though this is ultimately destroyed it does not become generally infiltrated with inflammatory cells; it also destroys the alveolar bone. It would seem, therefore, more rational and in keeping with both clinical observations and pathological findings to designate it by a name in keeping with these facts; we therefore refer to it by the name introduced in the sixth edition of this book as Parodontal Disease, meaning: disease beside or beyond a tooth.

Descriptions of the disease will be found in the works of Ambrose Paré, Fauchard, Toire, John Hunter, and others. These authors, however, described the disease in its advanced stages, the initial stages not having been recognised until recent years. This disease is widely distributed throughout the human race, and is also met with in animals under domestication, and wild animals, more especially those kept in captivity.

Various names have been given to the condition, *e.g.* Riggs' disease, pyorrhœa alveolaris, alveolar osteitis, etc.

(A) THE ANATOMY OF THE PARTS INVOLVED IN THE DISEASE.

Before a description is given of the morbid anatomy of parodontal disease, it is necessary to consider certain aspects of the normal anatomy of the parts involved in the disease in order that the pathological changes may be more readily appreciated.

(1) The Osseous Tissues.

As regards the mandible: fig. 611 shows that of a female, aged twenty-two, the alveolar process of which may be regarded as normal.

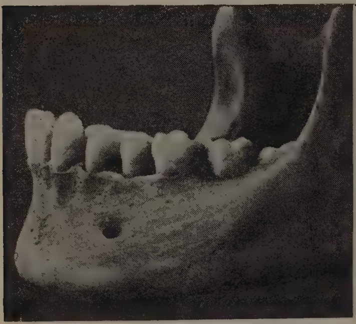

FIG. 611.—Mandible of female, aged twenty-two, with an alveolar process which may be regarded as normal.

The alveolar process and the teeth sockets are everywhere lined with a fairly dense layer of bone which appears in the radiograms (fig. 612) as a dark line. On the buccal aspects in the premolar and molar region the margin of the bone is close below the necks of the teeth, and, stretching across from one tooth to another in a straight line, forms the base of a definite triangular space between the approximal aspects of the teeth. In the incisor region the bone does not follow a straight line, but is continued upwards, so that the space between the incisors is considerably less than between the premolars and molars. The margin of the bone presents a regular outline, and the surface of the bone, which is smooth, is pierced here and there by nutrient canals. The appearance of the bone in radiograms is shown in fig. 612. Between the anterior teeth the bone has the appearance of a sharp spine, but it passes in a straight line between the posterior teeth. The outer aspect of the bone shows a dense layer, the inner part presenting a cancellous appearance. In the maxilla a similar condition obtains.[1] As age advances the alveolar process tends gradually to disappear.

[1] The radiogram of the incisors shows a slight break in the compact tissue towards the apex of the spine, but this is due to slight fracture (post-mortem).

(2) The Soft Tissues.

In healthy mouths, as age advances, the gum margin recedes as the alveolar process disappears, but a layer of compact bone is constantly present over the alveolar process. The mucoperiosteum should be firm and pale pink in colour. It should blend with the periodontal membrane at the neck of the tooth, the junction being distinguishable by the greater density in the character of the fibrous tissue. This band of tissue has been termed by G. V. Black the *dental ligament*.

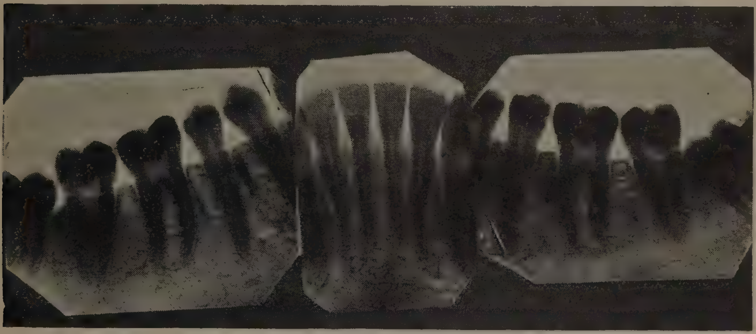

FIG. 612.—Radiograms showing the appearance of the bone in the mandible in fig. 611.

Though in the first place the gum may be attached at its margin and in continuity with Nasmyth's membrane, it is rare to find it so. J. G. Turner,[1] however, has shown that interstitially there may be no sulcus, the gum epithelium being prolonged on to the tooth crown and fusing with Nasmyth's membrane (fig. 613); in other cases there is the possibility that there is a shallow sulcus only, but not at the neck of the tooth, but halfway up its crown (fig. 614), while in others there was a potential rather than an actual sulcus (fig. 615). Clinically, however, the gingival margin is not attached to the tooth, as the gum falls slightly before reaching the tooth and forms a slight depression around the tooth. This depression is termed the 'gingival trough,' and is normally 0·5 to 1 mm. in depth. The floor of the depression is at the neck of the tooth, and consequently with the eruption of the tooth the depth of the depression decreases.

[1] *Brit. Med. Journ.*, June 4, 1927.

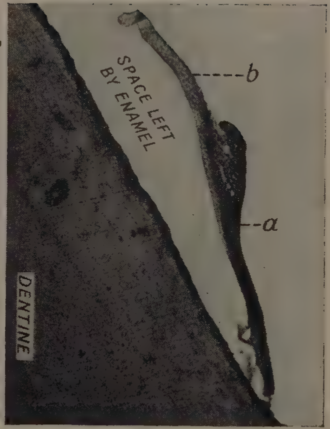

FIG. 613. — From a deciduous molar, patient aged 12. *a*, Gum epithelium deeply stained. *b*, Nasmyth's membrane staining badly. (J. G. Turner.)

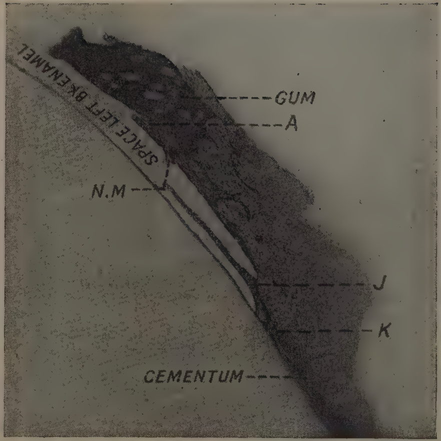

FIG. 614.—From a partly erupted permanent molar. N M, Nasmyth's membrane broken in sectioning. The apex of the sulcus was at A, well up the crown of the tooth. K, Epithelial prolongation along root. J, Junction of gum epithelium with remains of outer portion of enamel organ and Nasmyth's membrane. (J. G. Turner.)

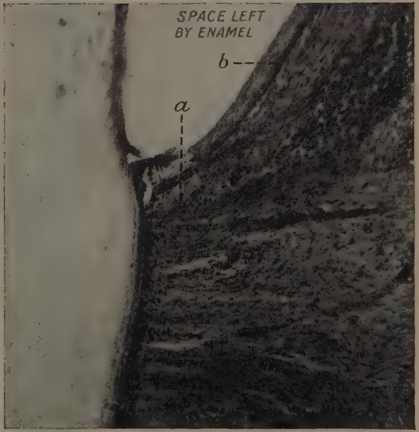

FIG. 615.—Deciduous canine, patient aged 7. The gum epithelium is attached to the tooth, but between it and Nasmyth's membrane is a line of demarcation (*b*) along which splitting can readily occur. There is thus a potential sulcus. (J. G. Turner.)

In other respects the minute anatomy of the gingival margins under strictly normal circumstances is as follows: The epithelium covering it is of varying thickness and covered superficially with several layers of flattened keratinised cells, deep to this the stratified epithelium shows a gradual sequence from squamous to cuboidal cells; on its deep aspect there are many interpapillary processes, sometimes branched and sometimes looping over to join other similar processes; usually the epithelial covering is described as being made up of four layers, the stratum corneum or keratinised layer, the stratum lucidum immediately under it, the stratum granulosum or granular layer, and the rete Malpighi, which is the deepest layer and composed of slightly differentiated cells, at times approaching a columnar or cuboidal type. The epithelium is continued down to the bottom of the gingival trough till it comes into contact with the tooth (fig. 616). Its nourishment is derived from capillary loops running in the connective tissue papillary processes (fig. 617). E. W. Fish[1] in a study of the microscopical anatomy states: 'It is impossible to find any piece of gum from the post-mortem room which is histologically free from the evidence of toxic absorption, even in small children,' and had recourse to tissue from a monkey (fig. 616) to demonstrate what must be regarded as strictly normal.

The functions of the gingival margins and gingival trough have never been satisfactorily demonstrated, but it is possible that they are for the protection of the periodontal membrane.[2] The free margin of the gum probably prevents direct injury of the membrane during mastication. With regard to the gingival trough, W. J. Penfold has shown that in the normal mouth the base of the trough is sterile. Mendel[3] observed that leucocytes are present in the gingival trough in healthy mouths, and this would seem to suggest that the leucocytes play the part of phagocytes in freeing the gingival trough from organisms and so indirectly protecting the periodontal membrane from infection.

The lymphatic system of the gingivæ, alveolar bone and tooth pulp has been traced in monkeys, dogs, cats and guinea-pigs by A. MacGregor[4] by the injection or absorption of solutions

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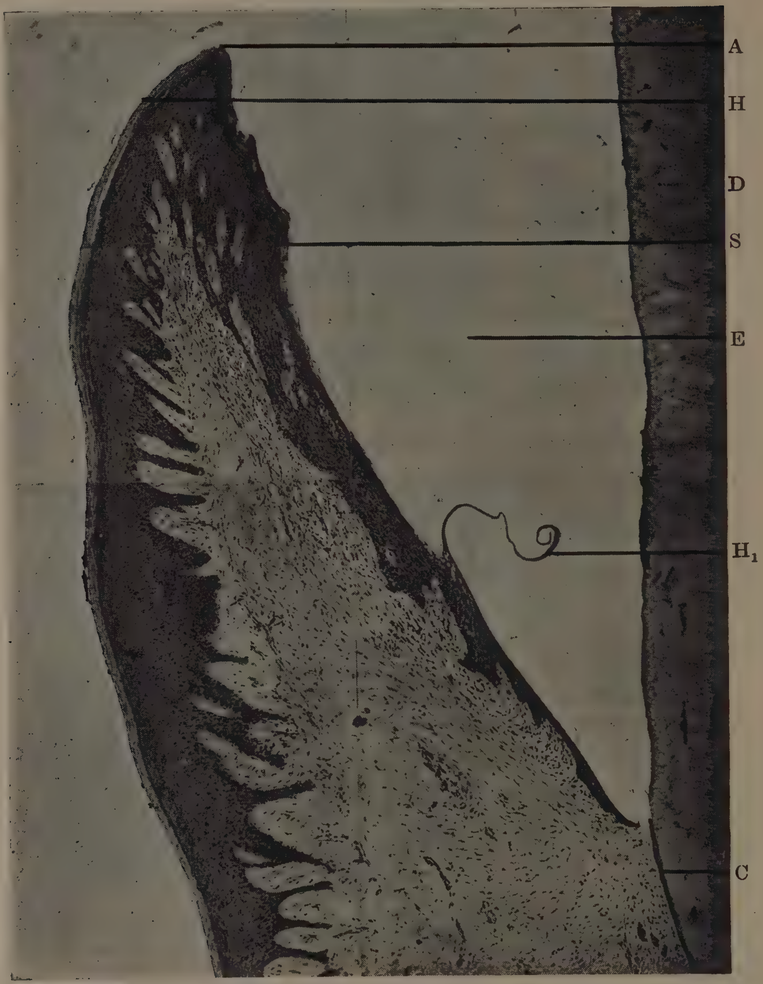
[1] *Brit. Dent. Journ.*, 1935, June 1 and 15.

[2] The gingival margin is comparable to the epitrichial margin of the epidermis at the base of a digital nail, which is normally in adherent continuity with the eponychium and nail; convention rules that this should be pressed back, and thus creates an 'epitrichial trough.'

[3] *Comptes rendus Soc. Biol.*, 1916, 587.

[4] *Proc. Roy. Soc. Med.*, 1936, xxix, 1237.



A = Apex of the gum margin.
D = Dentine.
H = Horny cuticle.
S = Bottom of periodontal sulcus or pocket.
C = Cementum.
E = Enamel (absent).

FIG. 616.—Healthy gum margin of a monkey. There is an unbroken horny cuticle all round the gum margin and a normal sequence in the stratified epithelium from cuboid to squamous cells. The underlying connective tissue is practically free from round-celled infiltration. The gingival trough is 0·25 mm. deep. The stripping back of a keratinised layer of the epithelial attachment (H_1) is an artefact; it was originally attached up to the point S. Although the epithelial attachment is thicker than is observed in some specimens there is no pathological proliferation and no downgrowth beyond the commencement of the cementum. (× 63.) (E. W. Fish.) (From *Brit. Dent. Journ.*)

of 10 per cent. to 20 per cent. basic lead acetate, the lead then being precipitated by the action of sulphuretted hydrogen as insoluble lead sulphide and the tissues then decalcified and sectioned. If we take the monkey as being probably most representative of conditions in man, the following are among MacGregor's findings :—

Lymphatic vessels are present in the periodontal membrane, and without pressure lead acetate solution was found to have travelled down the periodontal membrane from the gingival trough in these vessels, and into the lacunæ and alveoli of the alveolar bone ; the dentine and cementum were also reached, and though the path by which these tissues were reached was not clear, it is possible that it may have been via the cementum (fig. 618).

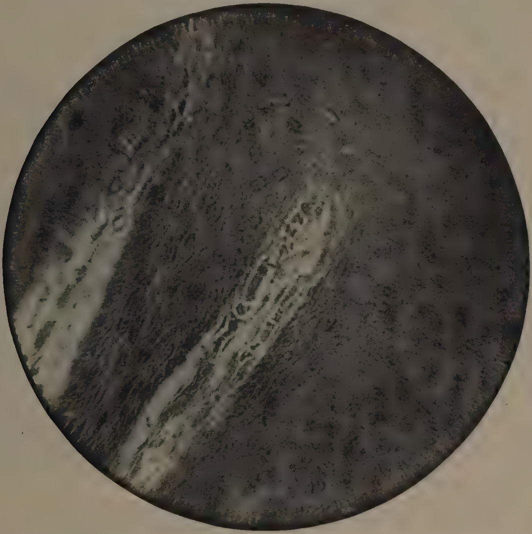


Fig. 617.—Section showing capillary loop at the extremity of a papillary process. (Box.)
(The Canadian Dental Research Foundation.)

Other conclusions concerning the lymphatic system of the jaws and teeth were that : 'drainage from the tissues of the upper jaw takes place mainly to the antero-lateral submaxillary lymph node, and thence to the supraclavicular group of lymph nodes. Drainage from the tissues of the lower jaw takes place mainly to the submental group of lymph nodes and thence to the paratracheal group of lymph nodes. There is a very large amount of anastomosis between the vessels and nodes of the lymphatic system in this region. Cross-anastomosis to the opposite side is a common phenomenon, particularly in the lower jaw.' Another important conclusion reached by this investigator is that: 'It may therefore be well, before considering the treatment of a pulpless or infected tooth by apicectomy or other operation, to make certain that the gingival tissues are healthy, and that they are kept in a healthy condition subsequent to the operation.'

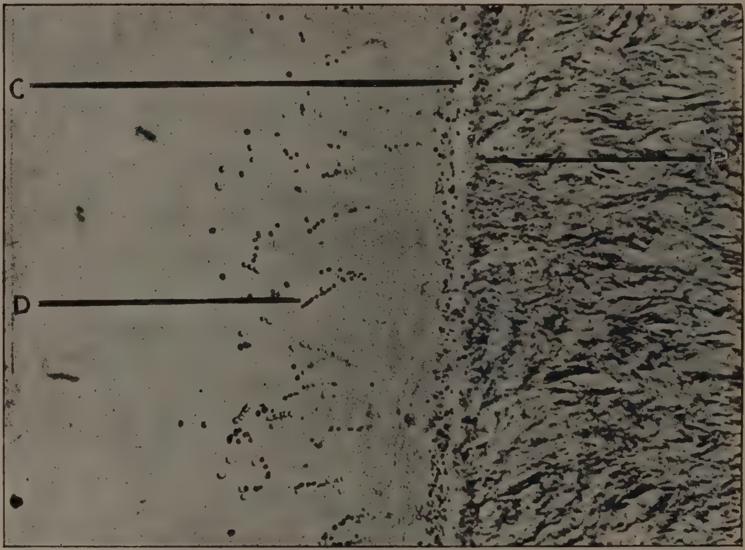


Fig. 618.—Dog. Lead acetate solution placed on a pledget of wool in an incision in the base of the gingival trough has travelled down the periodontal membrane, in which it can be seen at (P) and has reached the dentinal tubules (D) and cementum (C) of the tooth. (× 250.) (A. MacGregor.) (From *Proc. Roy. Soc. Med.*)

(B) CLINICAL APPEARANCES AND CLASSIFICATION.

In a young adult whose mouth is in a normal condition, the gums fit closely around the necks of the teeth and the spaces between the teeth are filled with processes of gum. Commonly, in the earliest stage of parodontal disease these processes of gum become slightly congested, and, if a probe is passed into the inter-proximal spaces, it will be found that the gum has been partially destroyed. The congestion gradually extends to the whole of the margins of the gums, which then bleed readily. Next, the processes of gum between the teeth gradually disappear and the normal festoons are obliterated. At this stage an examination of the teeth will usually disclose the presence of particles of food and other debris in the approximal spaces and small nodules of calculus on the teeth. Lastly, the inflammatory process involves the bone which, together with the periodontal membrane, is slowly destroyed. The gums recede, but the recession does not ordinarily keep pace with the destruction of the alveolar process, and the result is that around the teeth deep pockets are formed in which pus and other morbid and extraneous material accumulate and aggravate the condition. The mucous membrane is now deeply congested, the free margins of the gums are swollen and bleed at the slightest touch. The roots of the teeth are more or less exposed, and are covered with a layer of hard greenish-brown (serumal) calculus; there is a fœtid discharge, and the breath has a repulsive odour which is due to indol-forming organisms. A sickly, sour odour of the breath is noticeable when yeasts and bacteria causing carbohydrate fermentation are present. The teeth are freely movable and may become so loose that they can easily be removed. If the disease is left untreated the teeth are lost one by one, and with the loss of the teeth the inflammatory process clears up.

The disease, especially in advanced cases, is usually associated with congestion of the tonsils and of the mucous membrane of the mouth and pharynx, and it is often accompanied by superficial glossitis. The patient experiences an unpleasant taste in the morning. There is frequently recurring hæmorrhage from the gums, which is swallowed during the day, but occasionally escapes from the mouth at night and stains the pillow. The last symptom deserves special attention, as there is some danger that it may be mistaken for hæmorrhage from the lungs.

The **clinical appearances of the disease vary considerably** with the general condition of the patient, the resistance of the tissues, and the hygiene and functional activity of the mouth. At one period the condition may be acute, with a free flow of pus from the teeth sockets and a rapid destruction of the tissue. This state may be evanescent and may be followed by a period during which the inflammation is more or less quiescent. In some cases the teeth are comparatively free from calcareous deposits; in others they are completely coated. The disease may be more active around the anterior than around the posterior teeth, or vice versa. Again, the destruction of tissue may be more active in one region of the mouth than in another, *e.g.* the destructive process is often more advanced on the palatal than on the labial aspects of the incisors and canines.

In severe cases the disease may spread in the maxilla to the maxillary sinus, or in the mandible to the body of the bone.

'The buccal mucous membrane in advanced cases shows a number of small white stellate points, somewhat hard and shotty, lying underneath the mucous membrane, and corresponding in position to the site of the disease of the gum margin. From these glands bacteria of various species, generally bacilli, may be obtained.' (Goadby.)

In some cases, where care is taken by the patient to keep the mouth clean, the gums are not sufficiently abnormal in colour to attract attention, and the looseness of the teeth is the only indication of the presence of the disease. In such cases the probe will disclose deep pockets, and pressure on the gum will cause a discharge from the tooth sockets. In a few cases the disease is slow in its progress, and the surrounding bone actively reacts to the injuries caused by the toxic products on the sockets. This is well seen in the case shown in fig. 619.

In certain cases there seems to be a tendency for granulation tissue to form, causing the condition to simulate sarcoma. In one case under observation, associated with myxœdema, the gums were friable and presented a translucent appearance.

The disease may often be well advanced without the patient having become conscious that anything is wrong, the only symptom being a tendency for the gums to bleed readily, and this is often unheeded by the patient. With the formation of spaces between the posterior teeth, neuralgia may arise from septic infection. In the later stages there is often persistent neuralgia owing to the main trunks of the nerves being involved in the diseased process. Where pus formation is active the patient will often complain of a general itching of the gums.

It has been thought that the disease can be transmitted from one individual to another, but the balance of evidence is against it.

On account of the varying clinical appearances of this disease there have been *attempts to classify* it into different types, and in the many classifications which have been put forward there seems to be some agreement that there are two chief types :—

(i) Local, and starting as a marginal gingivitis ;
(ii) Systemic, and commencing primarily with atrophy of alveolar bone.

The former is by far the commoner and is almost universally acknowledged to start as a chronic gingivitis. The latter is said to be associated with malnutrition (see p. 616), endocrine disturbances, diabetes, and pregnancy. In this type the loss of alveolar bone may affect one, several, or all of the teeth and occurs generally, not necessarily starting and progressing from the alveolar crest ; it may only involve the apical regions of the teeth, the cementum of which may show evidence of resorption in the affected area. In its later stages the clinical appearances are indistinguishable from those of Type (i), and the treatment, apart from that of any systemic disability which may be present, is along the same lines.

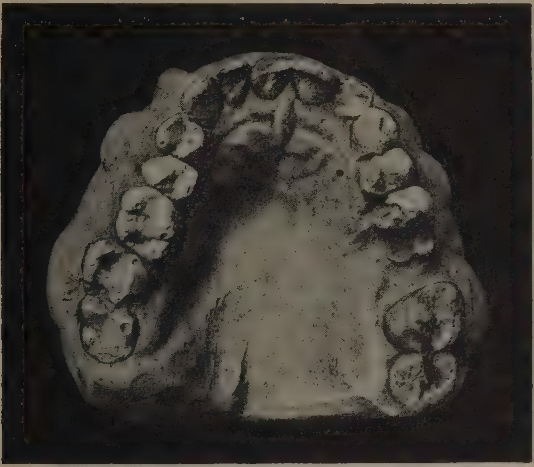


Fig. 619.—From a case under the care of J. G. Turner. Compare with fig. 643.

Clinical appearances alone are not altogether satisfactory guides as to the extent of the disease, and it is only by the aid of radiograms that the amount of the bone destruction can be estimated with any degree of accuracy. The following cases will serve to demonstrate the amount of bone destruction in relation to the clinical appearances.

The case shown in fig. 620 depicts an early stage of the disease. Clinically there is no recession of the gums beyond a partial disappearance of the interdental papillæ between the mandibular incisors. There was a marked marginal gingivitis, together with a congestion of the whole of the gums. There was a free discharge from the pockets around the teeth. This patient had a well-developed arch and there was marked attrition. The radiograms show that the alveolar process is already involved and that the destruction of bone has advanced to a considerable extent in the mandibular incisor region (fig. 621).

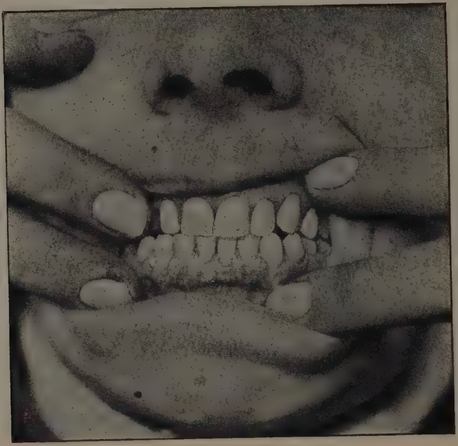


Fig. 620.

In the case shown in fig. 622 the clinical appearances suggest that the disease is more advanced than in the preceding case. The muco-periosteum was more swollen and congested. The interdental papillæ had quite disappeared, but the gum margin on the labial aspects of the teeth just failed to cover the necks of the teeth. The radiograms (fig. 623) show that the bone destruction was much more advanced than the gum recession.

Figs. 624 and 625 illustrate an advanced condition. The radiograms show the extensive destruction of the teeth sockets.

Those marked (*a*) and (*b*) also illustrate a point, namely, that the calculus may only extend to just under cover of the gum margin.

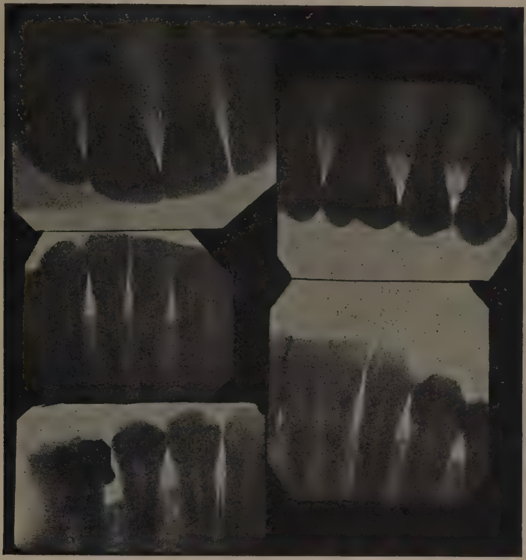


Fig. 621.—Radiograms of the case shown in fig. 620.

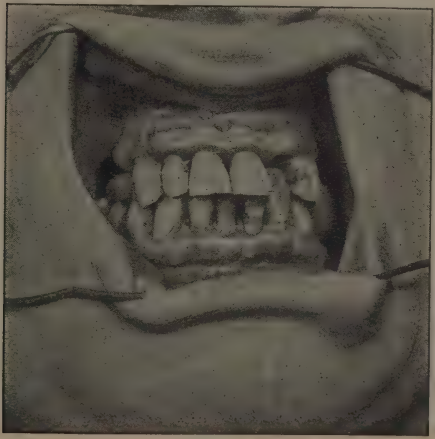


Fig. 622.

Rarefying osteitis around the roots of a maxillary molar is shown at (*c*) fig. 625. This case illustrates some features which are of interest from the point of view of prognosis. The patient, a man aged thirty-four, was a compositor by trade, and was subject to dyspepsia. Beyond a well-defined marginal gingivitis the gums were normal in colour and firm in consistency. The teeth were

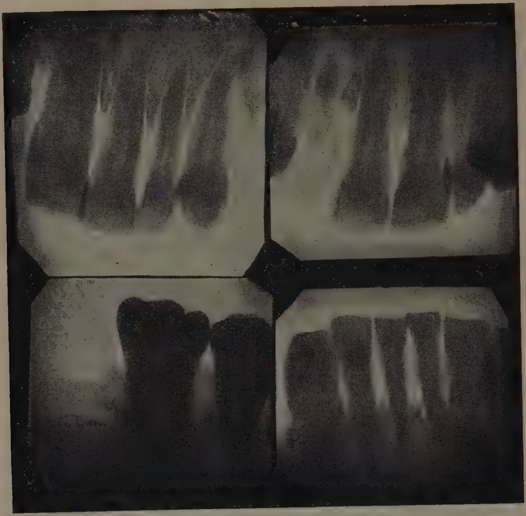


Fig. 623.—Radiograms of the case shown in fig. 622.

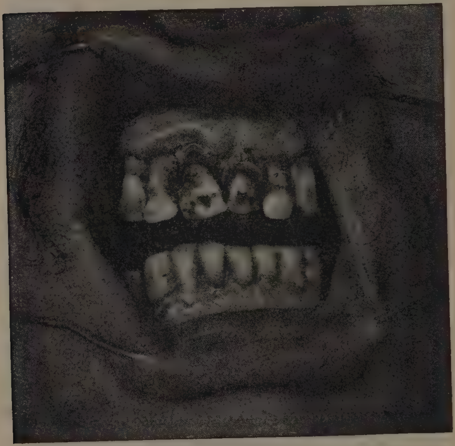


Fig. 624.

smothered with calculus; the gum margin was above the necks of the teeth, and the interdental papillæ had disappeared.

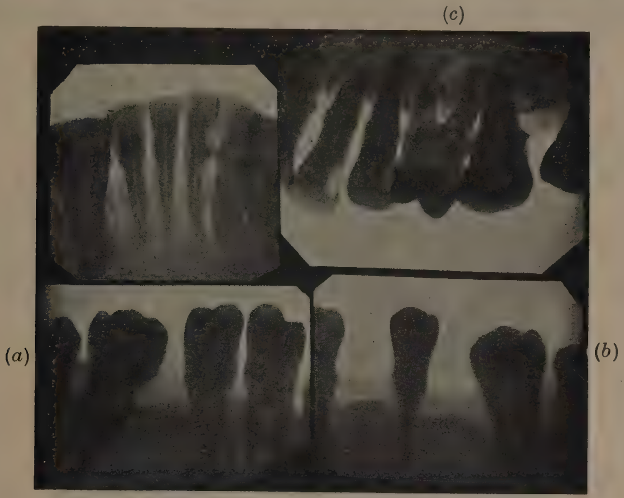


Fig. 625.—Radiograms of the case shown in fig. 624.

In the case shown in fig. 626 the patient was a mouth-breather and complained of bleeding from the region of the mandibular incisors. The teeth had been regularly cleaned. The gums were well up to the level of the necks of the teeth, and the inter-

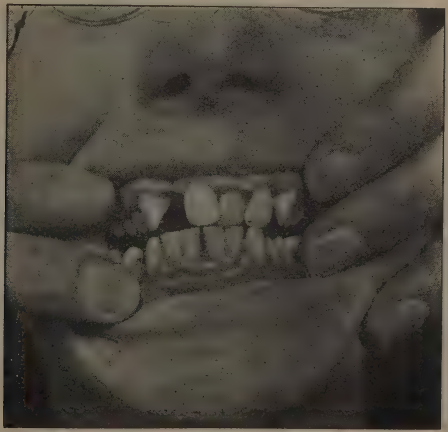


Fig. 626.

dental papillæ were not destroyed. There was marked gingivitis around the mandibular incisors where pus was present. An examination with a probe showed that there was extensive

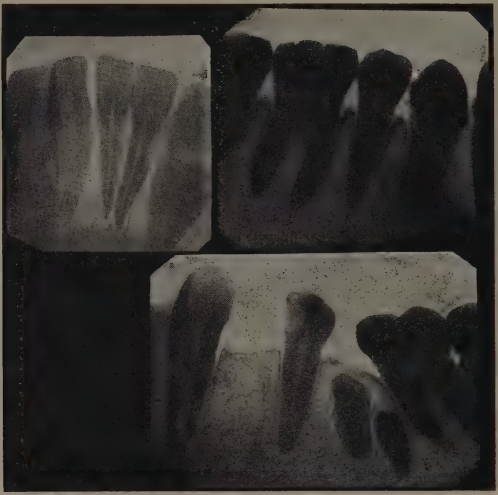


Fig. 627.—Radiograms of the case shown in fig. 626.

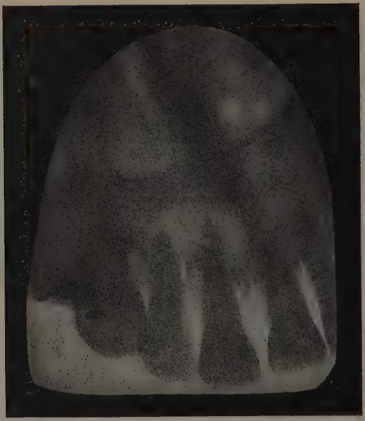


Fig. 628.

destruction of the alveolar process in the region of the mandibular incisors, while in the rest of the mouth the bone was unaffected.

In the case shown in fig. 628 the alveolar process on the medial aspect of the second incisor has been completely destroyed, and infection of the bone around the apices of the first and second incisors has followed. This patient had a well-defined parodontal abscess, with the pulps of the teeth alive.

(C) MORBID ANATOMY.

(1) The Alveolar Process.

Clinical experience indicates that parodontal disease usually originates either in the molar region or around the incisors. When it originates around the posterior teeth, the initial lesion in the bone is a slight destruction of tissue in the centre of the interproximal spaces as shown in fig. 629, a specimen which has the appearance of being normal unless carefully scrutinised. The disappearance of the layer of dense tissue and the breach into the cancellous bone indicate that the destruction of the bone has already commenced (fig. 630). As the destruction of the bone

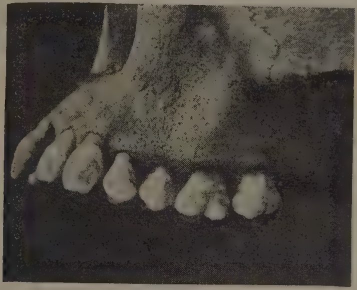


Fig. 629.

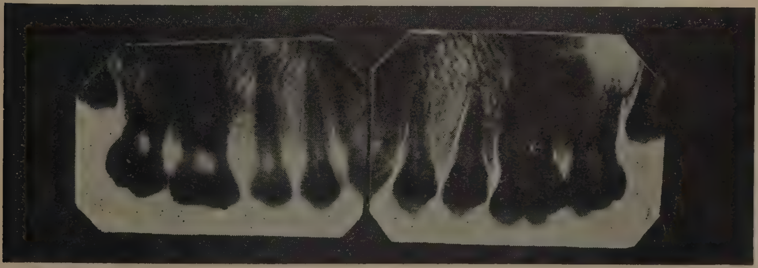


Fig. 630.—Radiograms of the specimen shown in fig. 629. Note the slight destruction of the bone in the centre of the interproximal spaces.

proceeds the outer and inner plates of the alveolar process become involved, but the loss of tissue, as a rule, is greater between the teeth than on the buccal and palatal aspects. This stage is shown in fig. 631. It will be observed that :—

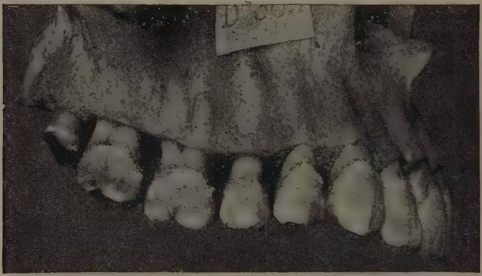


FIG. 631.—Maxilla in which the destruction of the bone has advanced beyond the stage shown in fig. 629.

(*a*) The destruction of bone around the anterior teeth is considerably less than around the premolars and molars.

(*b*) The loss of tissue around the molars is greater than around the premolars.

A further stage of the disease is shown in fig. 632. The bone has so far disappeared as to expose about half of the roots of the incisors and two-thirds of the roots of the molars. Around the canine and premolars the bone destruction is less advanced.

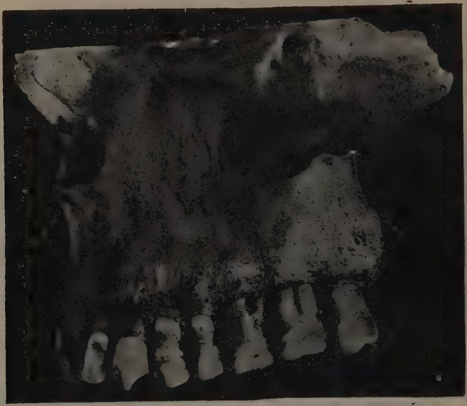


FIG. 632.—Maxilla showing considerable destruction of the alveolar process.

A specimen in which the disease probably commenced around the incisor teeth is shown in fig. 633. There is some loss of bone between the teeth, and the roots on the labial aspects are uncovered. The margin of the bone shows the pitted, irregular appearance characteristic of rarefying osteitis. It will be observed that there has been less destruction of bone between the first incisors than between the other anterior teeth. An examination of the specimen shows that around the incisors the destruction of bone is more advanced than around the molar teeth. The radiograms (fig. 634) bring out clearly the extent of the bone lesion and also the resistance of the tissues between the first incisors.

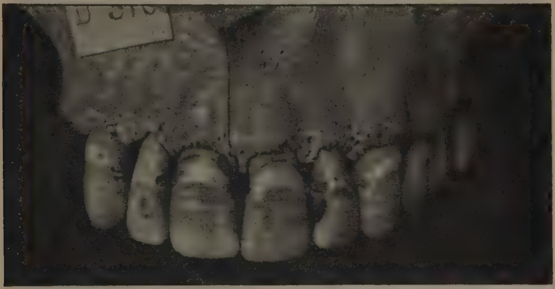


FIG. 633.—Specimen in which the disease probably commenced around the incisors.

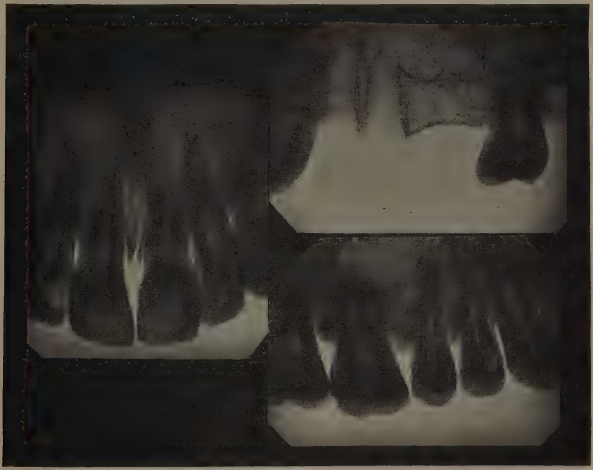


FIG. 634.—Radiograms of specimens shown in fig. 633. The extent of the bone lesion and the resistance of the tissues between the first incisors is clearly shown.

The specimen shown in fig. 635, from a male, aged thirty, is an excellent example of the morbid anatomy of an advanced case. The bone around the incisors has almost disappeared, and the molar teeth are uncovered nearly to their apices. Around the teeth are cup-shaped spaces which are due to the fact that the bone immediately adjacent to the tooth disappears first. The

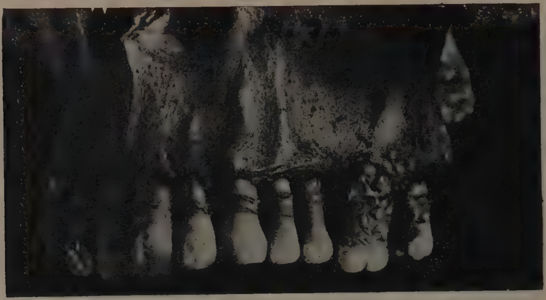


FIG. 635.—This specimen from a male, aged thirty, shows the morbid anatomy of an advanced case of parodontal disease.

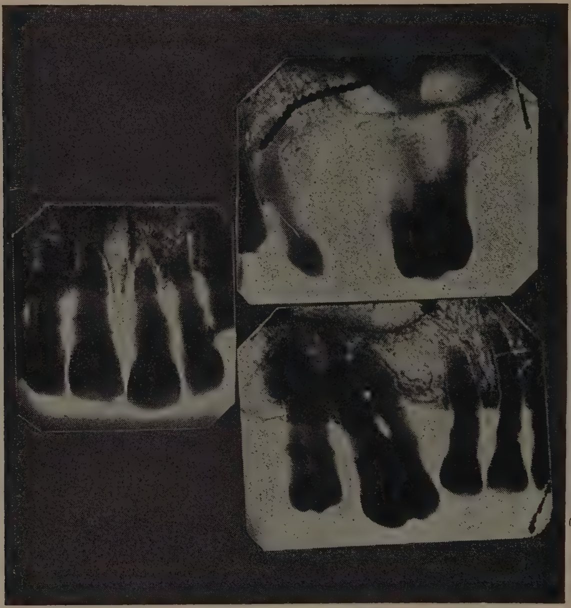


FIG. 636.—Radiograms of maxillæ shown in fig. 635.

appearance of the bone shows that there has been a marked rarefying osteitis. Nodular deposits of calculus are present on the teeth, and it will be observed that these are, in the case of the incisors, canines, and premolars, well away from the margin of the bone. The radiograms of this specimen are shown in fig. 636. The cup-shaped absorption of the socket is clearly shown in the right first molar of the specimen illustrated in fig. 637. Around the second molar the destruction of the bone has been of a chronic type, the cup-shaped absorption being absent. The radiogram of these two teeth (fig. 638) shows a very distinct difference in their appearance. Around the roots of the first molar there is

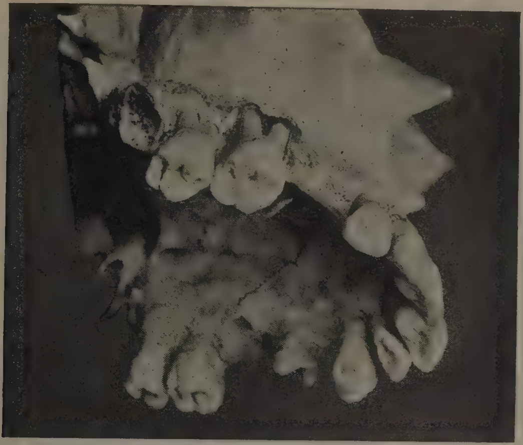


FIG. 637.—Maxilla showing cup-shaped absorption of the bone around the right first molar.

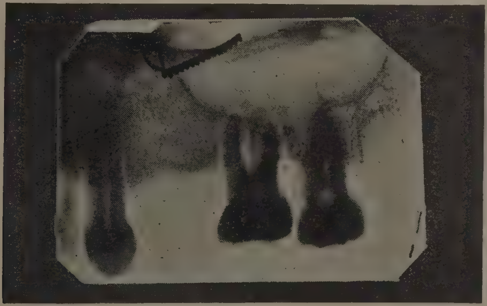


FIG. 638.—Radiogram of the first and second molars in fig. 637, showing the different degrees of bone destruction around these teeth.

a definite clear area, and the dense layer which marks the outline of the normal socket is absent. The bone around the tooth shows definite signs of rarefying osteitis. The bone bordering the roots of the second molar still retains the dense appearance, and this indicates that the rarefying process has not spread deeply into the bone around the tooth.

In the final stage of the disease the teeth fall out owing to the extent of bone destruction. The specimen shown in fig. 639 is an excellent example of this condition. The premolars and molars have fallen out and the alveolar process has entirely disappeared.

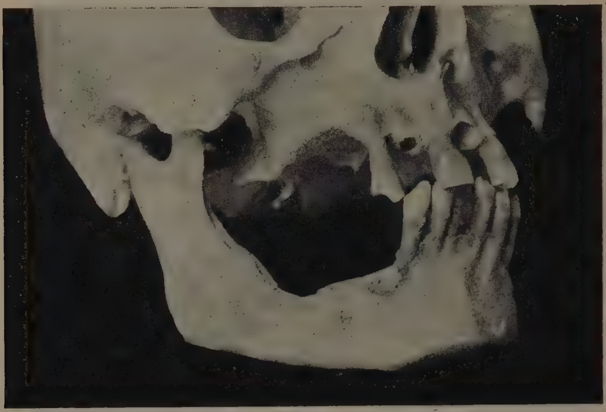


Fig. 639.—Final stage of the disease. The teeth have fallen out owing to the extensive destruction of the surrounding bone.

The condition of the mandibular canine shows how the final loss of the tooth is brought about. In life this advanced stage of the disease is denoted by the absence of 'ridges.'

In specimens showing marked attrition of the teeth the outer aspect of the alveolar process is often sclerosed and the rarefaction limited to the alveolar margins. A case of this type is shown in fig. 640. The radiograms (fig. 641) show that the rarefying process is almost entirely limited to the surface and has not spread deeply into the sockets. Compare the radiographic appearance of the bone around the incisors in this specimen and in the specimen shown in fig. 635. The marked attrition of the teeth indicates that the function of mastication has been efficiently performed, and the bone around the teeth, having therefore been kept well nourished with a plentiful flow of blood, was in a favourable condition to react to injury.

A specimen in which there is greater sclerosing of the bone is

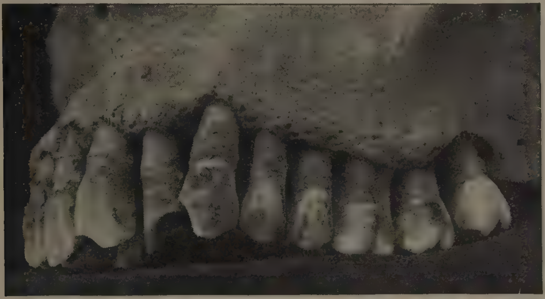


Fig. 640.—A specimen in which the bone shows marked reaction. Note the thickened margin of the alveolar process.

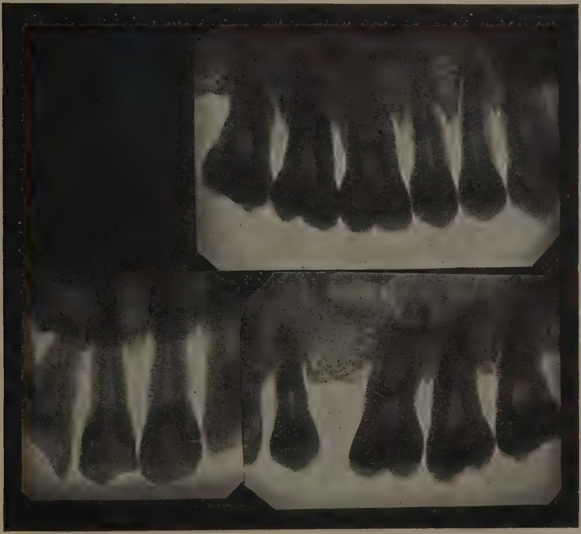


Fig. 641.—These radiograms of fig. 640 show that the rarefaction of bone is to a great extent limited to the surface.

shown in fig. 642. The position of the calculus in relation to the margin of the bone is evidence that well-defined pockets existed around the teeth. The surface of the bone around the teeth shows signs of a rarefying osteitis, but there is no cup-like absorption of the bone. The arch is well formed and the attrition of the teeth on the masticating surface points to good functional activity.

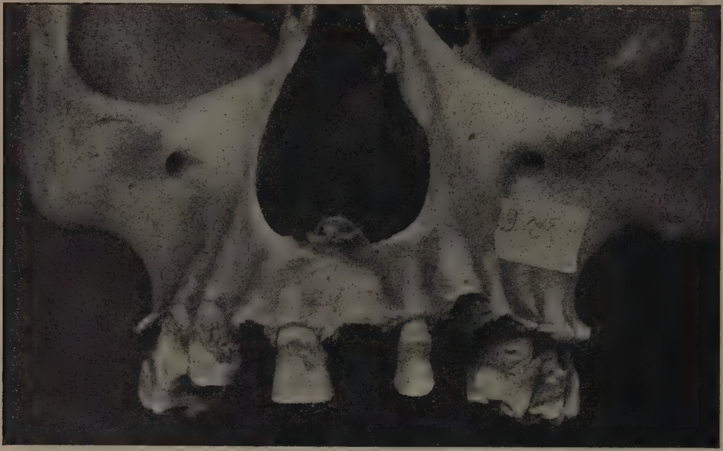


FIG. 642.—A specimen from a very chronic case showing considerable thickening of the margin of the bone.

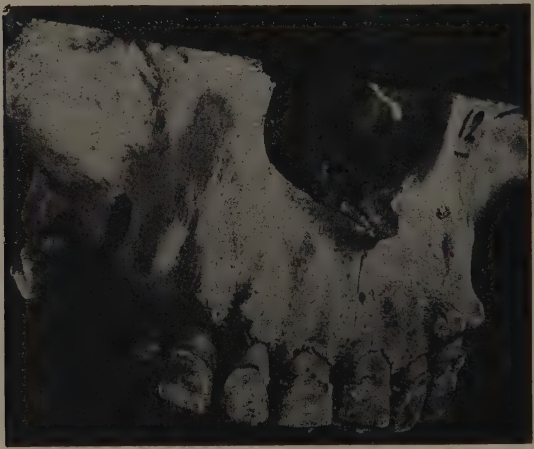


FIG. 643.—A specimen showing the formation of nodular masses on the outside of the alveolar process.

An example of the formation of nodular masses of bone is shown in fig. 643.

In practice a type of case is met with in which the bone destruction on the labial and palatal aspects of the teeth is much more advanced than on the approximal aspect. In many of these cases the gum margin is very little above the neck of the tooth, and a probe inserted between the gum and the tooth will pass almost to the end of the root. This condition is generally associated with the anterior teeth, and not infrequently with patients whose teeth are unduly prominent. A specimen illustrating the morbid anatomy of this type of case is shown in fig. 644. The bone on the labial aspects of the right canine and first incisor has completely disappeared; the left first incisor and canine are only covered by bone in the neighbourhood of the apices. Note the deposit of calculus around the necks of the teeth and the freedom from calculus of the remaining exposed portions of the roots. On examining the position of the teeth in relation to the bone, it will be seen that the arch is decidedly narrow and that the general direction of the teeth is too vertical.

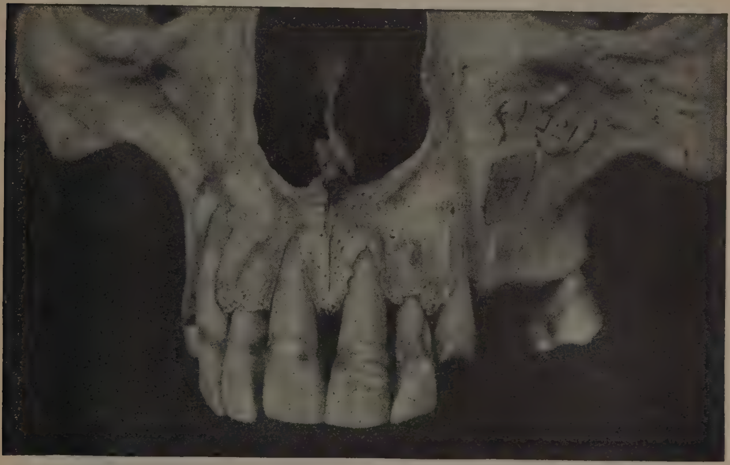


Fig. 644.—In this specimen the bone destruction is most marked on the anterior aspects of the teeth.

A point worthy of attention is that the disease spreads in the maxilla more actively and extensively than in the mandible; especially is this the case in the region of the molars. The more limited area of bone destruction in the mandible is due to the nature of the osseous tissues, the bone in the maxilla being of a much more cancellous character than in the mandible.

A mandible showing extensive destruction of the bone in the region of the molars is shown in fig. 645, and the radiograms of the specimen in fig. 646.

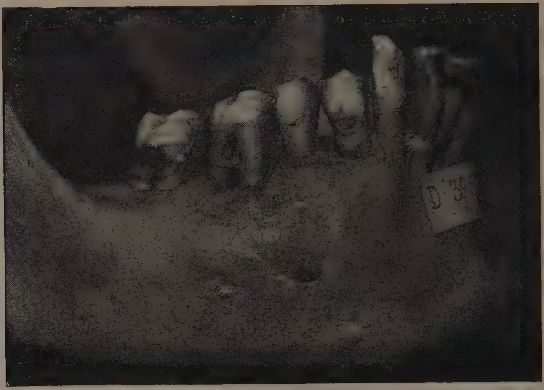


FIG. 645.—Mandible from an advanced case of parodontal disease in the region of the mandibular molars.

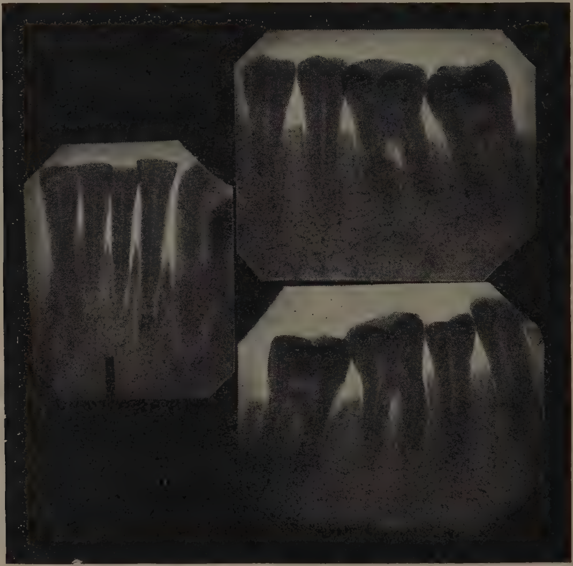
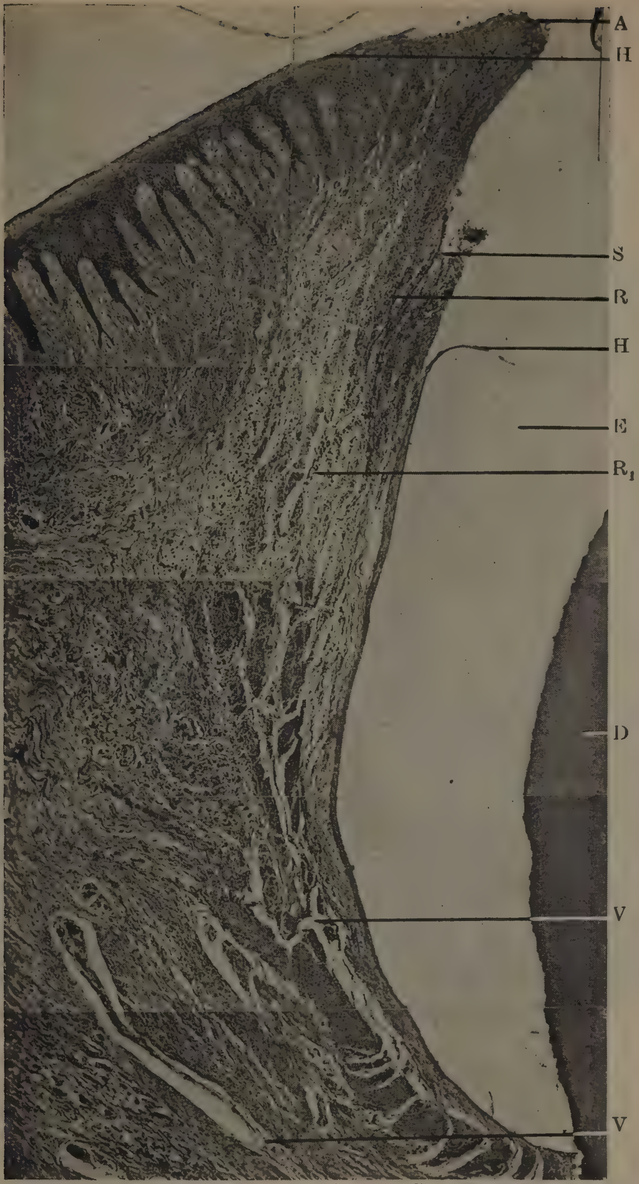


FIG. 646.—Radiograms of the specimen shown in fig. 645.

The series of specimens just described indicates that the bone lesion is a progressive rarefying osteitis which commences at the margin of the tooth socket and eventually destroys the bone until the tooth is shed owing to the loss of its attachments.



A = Apex of the gum margin.
C = Cementum.
D = Dentine.
E = Enamel (absent).
F = Fibres of the ligamentum circulare.
G = Downgrowth of epithelial attachment.
H = Horny cuticle.
M = Epithelium.
P = Pus.
R = Round-celled infiltration.
S = Bottom of periodontal sulcus or pocket.
T = Tartar.
V = Blood and lymph vessels.

(Keys to figs. 647 to 650 inclusive.)

FIG. 647.—Human gum margin from child aged 4 years. The apex of the gum margin has become bruised and the cuticle is scraped away. The sulcus, 0·25 mm. deep, shows a tear (ante-mortem) at the bottom. A round-celled infiltration of the adjacent corium has taken place and is spreading along the vessels to R_1. Otherwise the specimen is normal. There is no proliferation and no downgrowth of epithelium. (× 63.) (E. W. Fish.) (From *Brit. Dent. Journ.*).

(2) The Soft Parts.

The morbid anatomy of the soft parts concerned is well shown in the following illustrations taken from a paper by E. W. Fish [1]; these should be compared with fig. 616, a section from a monkey which may be regarded as normal. Fish has shown that the earliest sign that any pathological process is commencing is a reaction to injury showing in the connective tissue under the

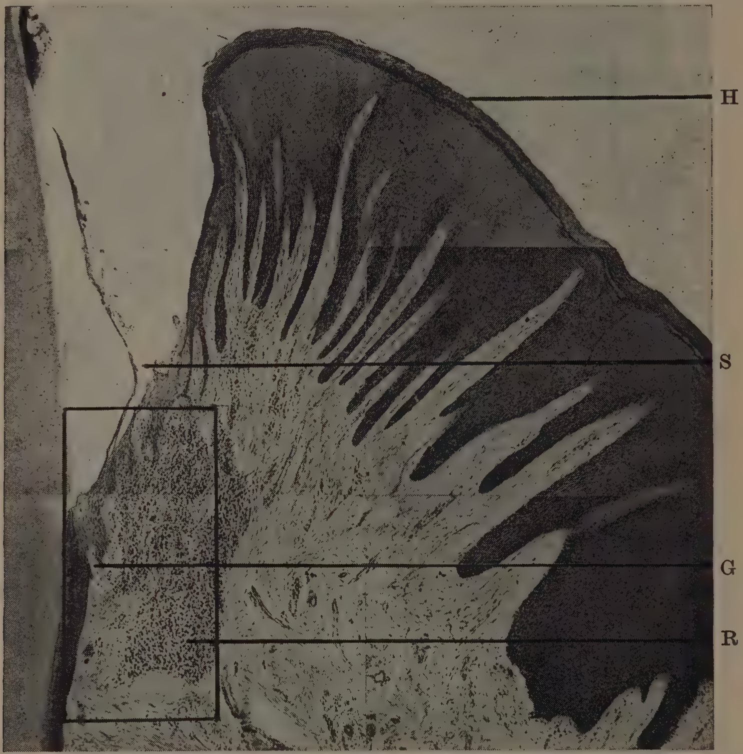


Fig. 648.—Human gum margin. The trauma in this case produced its maximum effect not at the apex of the gum margin but at the bottom of the sulcus (S) as shown by the tearing of the epithelium. This tear was ante-mortem and chronic, as indicated by the infiltration below it which is of the round-celled type. This infiltration has spread under the whole of the epithelial lining of the trough and surrounded the epithelial attachment. The former is beginning to proliferate into the corium and the latter downwards along the cementum, the stimulus to growth being the same irritant which determined the round-celled infiltration (*i.e.* autolytic products of trauma or chemical or bacterial intrusion). The gingival trough is about 0·5 mm. deep. (× 63.) Fig. 649 is a higher magnification of the enclosed portion. (E. W. Fish.) (From *Brit. Dent. Journ.*)

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[1] *Brit. Dent. Journ.*, 1935, June 1 and 15.

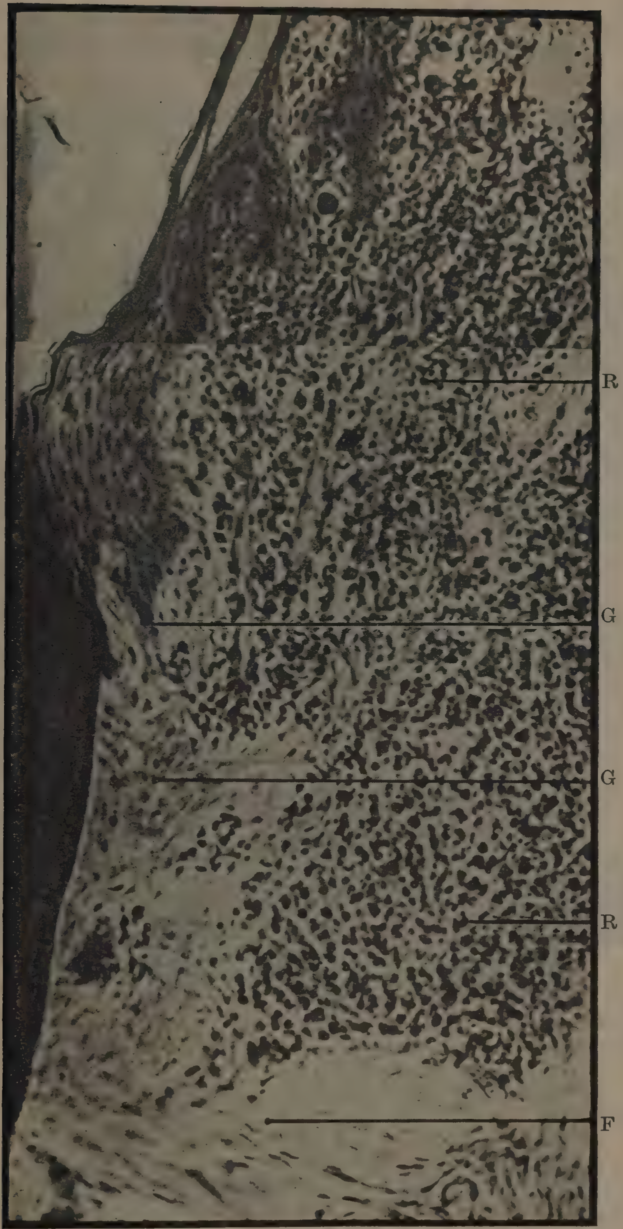

FIG. 649. Higher Magnification of Figure 648 showing the epithelial attachment beginning to grow downwards. Toxic substances are beginning to diffuse into the fibres of the ligamentum circulare (F) as shown by the presence of round cells. As these fibres become detached from the cementum the latter becomes contaminated. The epithelium then grows down to find healthy cementum to which it may attach itself. (× 192.) (E. W. Fish.)

epithelium lining the gingival trough as a small round-celled infiltration (fig. 647). This, in his opinion, is in response to mechanical damage to the gingival margin caused by the trauma of mastication, and the initial point of damage may show either at the actual gingival crest or in the depth of the trough. The inflammatory response naturally occurs at the point of injury

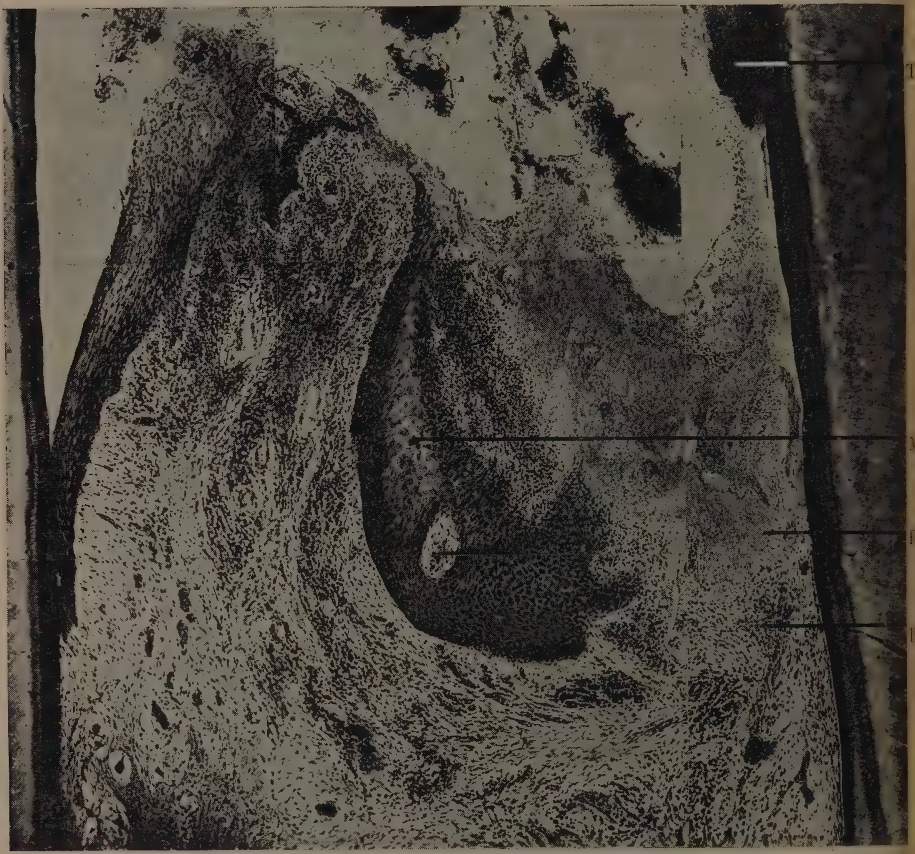

FIG. 650. Human interdental papilla. A late stage of the disease. On the right the epithelium, together with the epithelial attachment, has entirely disappeared and is replaced by a mass of round cells, parasites, pus and saprophytes, debris and tartar. On the left the epithelial attachment persists and is growing down towards healthier tissues. The central mass of epithelium is growing to no purpose, stimulated to do so by its toxic environment. (× 63.) (E. W. Fish.) (From *Brit. Dent. Journ.*)

and precedes any downgrowth of epithelium from the depth of the trough in contact with and adherent to the tooth.

The continuance of this downgrowth, by destroying the attachment of the periodontal fibres to the tooth, ultimately creates a 'pocket.' Figs. 648, 649 and 650 show how and where the initial lesions and reactions occur. Eventually the 'pocket' by extension to the apex of a tooth may cause its exfoliation.

Thickening and varicose changes have been described and figured in the pulpal and periodontal innervation in parodontal disease by R. V. Bradlaw.[1]

(3) The Teeth.

Teeth taken from cases of parodontal disease exhibit definite changes. The hard dental tissues may show absorption and the periodontal membrane may be thickened as the result of the chronic toxæmia, and this necessarily implies pathological changes in the adjacent bony tissue. The pulps of the affected teeth show a tendency to fibrosis.

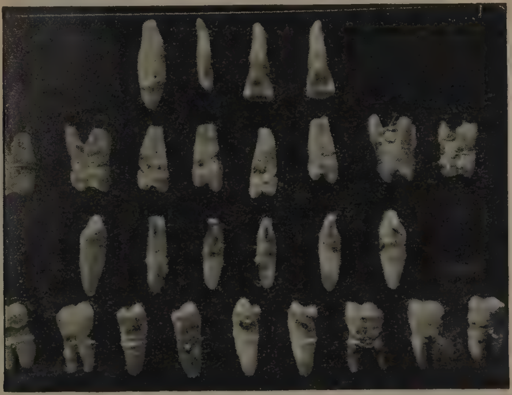

FIG. 651.

The degree of rarefying osteitis bears some relation to the rapidity of parodontal destruction, and facility for invasion of the tissues by toxic products increases in proportion to the amount of rarefying osteitis. The condition of the teeth in fig. 651 indicates that there was marked rarefying osteitis, and therefore facility for absorption of toxins, etc., into the blood-stream.

[1] *Proc. Roy. Soc. Med.*, 1939, xxxii, 1040.

Radiograms of the alveolar process of the patient are shown in figs. 652 to 654.

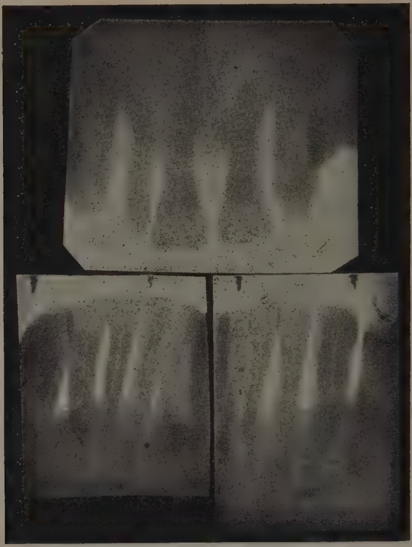

FIG. 652.

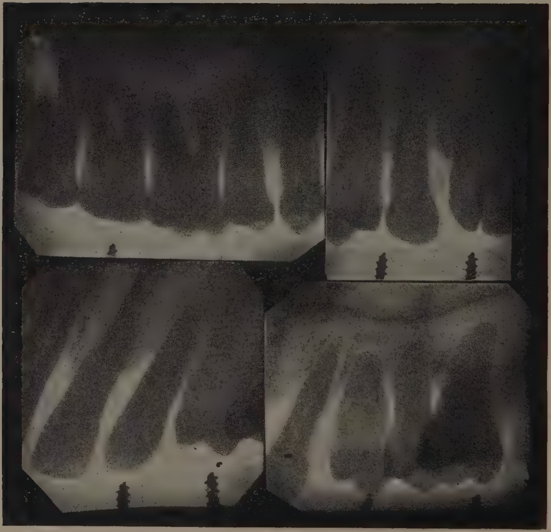

FIG. 653.

Large masses of adventitious tissue are frequently found in the cleft between the roots of the molar teeth, indicating extensive loss of osseous tissue at those points. The condition of the teeth in cases exhibiting deep pockets and profuse suppuration is well shown in the specimens illustrated in fig. 655.

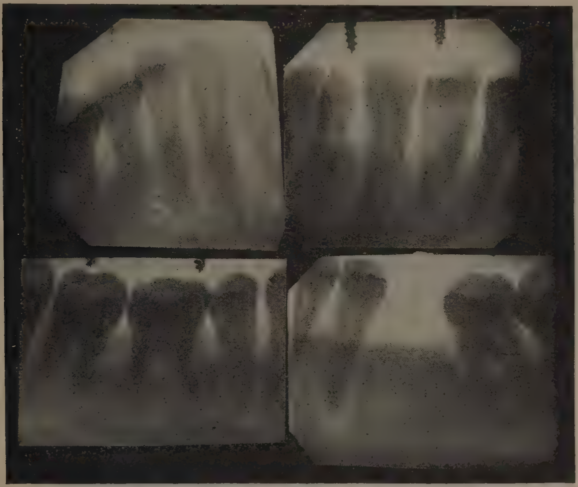

Fig. 654.

Fig. 655.

In cases where the disease has been extremely chronic the teeth become very brittle. The brittleness is due to increased calcification of the dentine and cementum, and results in their increased translucency, especially about the apices ; the process is as follows : The toxins injure the tooth tissues, which, like other tissues of the body, react, the reaction taking the form of increased calcification of the soft contents of the dentinal tubes and cemental lacunæ, thus rendering the tooth more brittle. If the teeth are immersed for six or eight weeks in borax carmine it will be found that the translucent area does not stain to the same extent as the other parts of the teeth, which indicates that the soft tissue normally present has undergone calcification.

Sections of teeth from three cases of parodontal disease are shown in fig. 656.

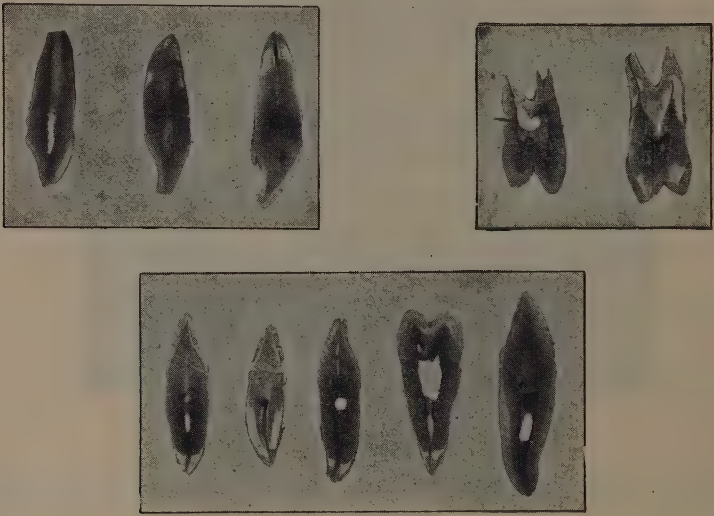

Fig. 656.

Teeth from cases of parodontal disease showing translucent areas.

(D) PATHOLOGY.

There are several important clinical facts in connection with the disease in man which may assist in the endeavour to trace the pathology of the disease. In *non-mouth-breathers* the disease commences in the molar region and gradually spreads to the anterior teeth, while in *mouth-breathers* the disease commences at an earlier date and often remains for some period limited to the anterior teeth. The commencement of the disease in the *non-mouth-breathers* occurs in the region where lodgment of food is liable to occur, and probably here the initial lesion is an injury to the gingivæ from food debris.

The fact that in *mouth-breathers* the disease commences in the incisor region is instructive. If the mouth of a child affected with mouth-breathing, but with functional molars, be examined, a gingivitis will be noticed limited to the incisor teeth, especially the mandibular ones. The absence of gingivitis in the molar region is due to the fact that the function of this part of the mouth is being properly performed ; the friction of mastication and possibly the rubbing of the buccal mucous membrane against the gums removes the superficial layers of epithelium, and stimulates a healthy reaction in the tissues. In the front of the mouth the normal friction of the lips against the gums is absent, the surface epithelium accumulates, together with the debris of food and micro-organisms, and injury results to the gingivæ. As long as mouth-breathing continues the gingivitis persists owing to the constant injury, and eventually the periodontal membrane becomes involved. There is little doubt, therefore, that, with mouth-breathers, the initial stage of the disease is a localised injury to the gingivæ of the anterior teeth.

In considering the pathology of this disease, due weight must be given to *the degree of resistance of the osseous tissue*, as it probably plays an important part in the rate at which the disease progresses. An examination of dried specimens certainly suggests that the more compact the osseous tissue the greater the barrier to the spread of the disease. The question then arises whether the tissues of the tooth socket are as resistant to disease as we might naturally expect them to be. Although no definite data bearing on this point exist, there are certain phenomena which tend to show that the tissues in many people lack normal resistance. It is an axiom that to maintain a part in health the function of that part must be maintained. There is little doubt that in modern times the teeth and surrounding tissues are not called upon to perform their normal functions, with the natural result that the tissues are deprived of a normal blood supply and consequently suffer in vitality. The bone surrounding the teeth is, as we know, a tissue of transient structure, depending for its existence on the teeth themselves. It is therefore reasonable to assume that there is a very close relationship between functional activity and the resistance of the tissues. With our present diet, functional activity is reduced because foods are so prepared that a very small amount of mastication is required. This loss of the function of the teeth must seriously affect the structure of the bone forming the alveolar process, and render it less resistant to attack. Clinical observation supports this view.

In mouths which show that the function of mastication is efficiently performed, the alveolar process is well developed, and if such mouths are attacked by parodontal disease the progress of the disease is not rapid. On the other hand, in individuals who masticate imperfectly, the bony coverings of the teeth are thin and sparse, and the disease when once started progresses rapidly. It seems probable, therefore, that imperfectly formed teeth sockets play an important part in the progress of the disease, inasmuch as their power of resistance is considerably diminished.

There is another point to be considered in connection with the resistance of the bone, viz. that *any inimical condition affecting the osseous system as a whole is likely to show itself in the alveolar process at an early age*, the reason being that the alveolar process is a transient structure.

It is at present controversial whether *adequate and efficient diet during the period of growth and development* of the parts involved influences their stability and resistance to infection. It is a fact that during this period certain materials are required to bring about their ideal development, and it seems logical to conclude that if these materials are inadequate or not available in assimilable form at the time they are required, the parts then forming will be faultily made and so, presumably, less able to withstand the stresses and infections to which they may be exposed. The writer[1] first saw the boys at a large charitable institution in 1907, and at that time the dietary deficiencies of many of them before admission had been appalling; they had not had the wherewithal or power to build either efficient bodies or teeth, and there were several boys there aged about fourteen or fifteen who had parodontal disease so badly that their permanent first molars were only attached by their extreme apices and were removed with fingers, and some of the anterior teeth had dropped out. There can be little doubt that the inherent vitality of the tissues concerned can be lessened in this manner.

The recent work of James and Counsell gives one a detailed picture of the progress of the pathological process and how the changes which occur are brought about. In their account they record that normally the gingival trough is non-existent, as the epithelium lining it is attached to the tooth substance and is, at the gingival crest, in continuity with Nasmyth's membrane; one has seen in sections of cats' teeth cut *in situ* that this is so with regard to these animals when young, but in man, at all events, the condition can only be regarded as transient, because clinically it is rare to observe it (see figs. 613 to 616).

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[1] *Brit. Dent. Journ.*, 1938, lxiv, 245. (E. S.)

With the commencement of irritation of this gingival margin, if not before, this epithelial attachment to the tooth ceases except at its deepest portion, and from now a gingival trough is always present. Following on this the deepest portion of the epithelium lining the trough proliferates on its deep aspect extending into the corium of the gum in contact with and adherent to the tooth, and at the same time there is a loss of epithelium from the superficial aspect of the deepest portion of the trough, so that the trough becomes exaggerated and what is clinically known as a ‘ pocket ’ is formed.

From the more recent work of E. W. Fish it appears that reaction in the form of a small round-celled infiltration precedes the downgrowth of epithelium.

This process is continuous and progressive and the loss of epithelium from the superficial aspect of the deepest portion of the pocket is always preceded by its proliferation on the deep aspect, which at this point maintains contact with and adherence to the tooth, whether the adjacent portion of tooth be enamel or cementum. In this way it will be gathered that however deep a pocket may be, it is always lined with epithelium, even to its depth, and that from its deepest point the epithelium is continued downward attached to the tooth for a short distance. This creeping downward of the epithelial attachment separates the periodontal membrane from its attachment to the tooth.

Concurrent with these happenings there is an inflammatory infiltration into the corium of the gum, the epithelium lining the pockets is also infiltrated, and that abrasions or breaches of this surface may occur is shown by the clinical sign of hæmorrhage. Serumal calculus is always found on the teeth opposite such abraded portions of the epithelial surface, and it is said that the *Leptothrix buccalis* is always present in this deposit.

As pockets are formed it necessarily follows that the so-called circular ligament of periodontal membrane around the neck of the tooth is destroyed ; it does not, however, cease to exist, as its function is taken up by other and deeper fibres of the periodontal membrane.

The periodontal membrane itself is not usually infiltrated with inflammatory cells to any great extent except in advanced or acute stages. The alveolar bone is first affected at its alveolar crest; this is progressively absorbed, though sometimes in cases where the teeth have been functioning efficiently, sclerosis occurs, as has been shown in the description of the morbid anatomy.

Thickening of arteries and dilatation of veins occurs in the parts affected as with other inflammatory processes, and the degree and amount of pus present depends on the type of organisms present and the resistance of the parts which they are attacking.

E. W. Fish considers that parodontal disease starts as a lymphangitis in the region of the gingival trough, and the possibility of this is of importance since organisms and their toxins travel mainly along the lymphatics. A. MacGregor,[1] too, has demonstrated the communications of the lymphatics of the gingival trough with those of the periodontal membrane, and that those of the periodontal membrane appear to be in communication with those of the spaces of alveolar bone and the pulp. This lymphatic communication, moreover, emphasises the importance of gingival cleanliness in the prognosis of many forms of conservative treatment of teeth, notably perhaps in the treatment of pulpless teeth, especially when there has been any periapical infection.

F. W. Broderick considers that a condition of alkalosis, by favouring the deposition of salivary salts on the teeth, predisposes to and ultimately, in conjunction with the other factors, brings about this condition. R. Champion's [2] work on salivary reactions, however, strongly indicates that the blood alkalinity has no effect whatever on the salivary *p*H, and seems rather to contra-indicate Broderick's conclusions.

The conclusions to be drawn from an examination of skulls showing the disease are :—

(*a*) That the bone lesion is a progressive rarefying osteitis commencing at the margin of the alveolar process, and (*b*) that the rate of destruction is in proportion to the density of the bone and its functional activity.

Znamensky [3] records the result of his examination of sections obtained from a female, aged thirty-nine, who had suffered from pyorrhœa alveolaris. The patient died of acute anæmia, the result of hæmorrhage after parturition. In all the sections a deposit of calculus was present under the gum margin. The sections showed that the pathological process was of an inflammatory character, originating in the gum, and then successively involving the periodontal membrane and the adjacent bone and finally causing their destruction. The destructive process in the bone near the margin of the socket consisted in the decalcification of the bone and its transformation first into an osteoid tissue and afterwards into a fibrous intervening tissue which subsequently became infiltrated with leucocytes. In the part of the socket where the bone-marrow was present lacunar absorption of the bone was seen. The whole process was in the nature of a rarefying inflammation.

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[1] *Proc. Roy. Soc. Med.*, 1936, xxix, 1237.

[2] *Brit. Dent. Journ.*, 1932, September 1.

[3] *Brit. Dent. Journ.*, xxiii, 585.

The view expounded in the foregoing pages, viz. that the disease starts at the gingival margin and gradually involves the tooth attachments, is not accepted by all writers. Gottlieb and Fleischmann[1] hold that atrophy of the bone is the primary causal factor in the etiology of the disease. They maintain that the sections shown in their paper afford evidence of destruction of the bone by autolysis before the small-celled infiltration of the gingival margin. Their sections, however, show that in the earliest stage the epithelium on the inner side of the gingival margin is thickened and that the epithelium pushes its way downwards between the cementum and the attachment of the periodontal membrane ; they also figure a small-celled infiltration in the sub-epithelial tissue which they regard as normal. But the fact that this small-celled infiltration is beneath the thickened epithelium is strongly suggestive of pathological changes—it is indeed the reaction of the mesoblastic tissue to the invading epithelium.

More recently Gottlieb has put forward the following as a tentative explanation of the pathology of what some regard as the *systemic type of parodontal disease* : the root surface of the tooth is damaged by toxins circulating in the blood and the attached periodontal fibres atrophy and are not replaced ; the existence of alveolar bone depends on stimuli transmitted to it via the periodontal membrane and Sharpey's fibres, and in the absence of these stimuli the alveolar bone atrophies and disappears. Following on this destruction of the periodontal membrane there is a downgrowth of epithelium from that covering the gum, causing the rapid but uneven development of deep pockets. The gums are not reddened, neither do they bleed much at first ;

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[1] *Dental Cosmos*, lxiii, 215.

at first there is little or no calculus, but it occurs later. The onset and progress are apt to be rapid.

T. B. Hartzell[1] considers that the lymphatic drainage of the tissue contiguous to the teeth is the most perfect in the body, and he states that the very method of attachment of the tooth in its socket increases the danger of general infection. 'The elastic[2] nature of the periodontal membrane makes it possible for the teeth to bear the shock of mastication, but the same elasticity favours the transmission of infection. If there is infectious material around the tooth, the strain of mastication forces the tooth downwards so that the root acts as a plunger, injecting the septic matter into the surrounding tissue.'

Hartzell's views as expressed above have recently received ample confirmation and proof by the work of Okell and Elliott[3] and the subsequent work of Round, Kirkpatrick and Hails.[4] The former of these workers examined bacteriologically the blood in 138 patients having both clean and dirty mouths, who were undergoing tooth extraction. Ten c.c of blood were taken immediately before extraction and another 10 c.c. within five minutes afterwards. It was found that 60·9 per cent. of all showed a *transient streptococcal bacteriæmia* after operation, but *of the mouths with marked gum disease* 75 *per cent. showed it.* The degree of bacteriæmia depended on the severity of the gum disease and the damage done at the operation. None of the streptococci recovered were of the hæmolytic variety, but were of the *S. viridans* type. The bacteriæmia was only of short duration, as was shown in ten cases where a positive result was obtained and a third specimen of blood taken from ten minutes to eight hours after operation, all of which were then sterile.

A most interesting finding was that of 100 of their cases tabulated as having 'marked' or 'moderate' gum disease, nine showed a streptococcal bacteriæmia *before* operation.

The work of Round and others followed; they sought the effects described by Hartzell, the 'pumping' effects of the teeth in parodontal disease during the act of mastication as a cause of bacteriæmia, by giving patients a 'mint lump' to chew for ten minutes. Blood for control purposes was taken beforehand and in all cases was negative on culture, and in the blood taken after the exercise *only those showing parodontal pocketing gave a positive result.* In one case they obtained a staphylococcus. Such leaks or escapes of non-pathogenic organisms into the circulation are, in a normal person, followed by their rapid removal by phagocytosis, but in a person having diseased or defective cardiac valves the organisms may be trapped there and precipitate a bacterial endocarditis, as the same type of organism is frequently isolated from definite cases of endocarditis as is obtained in cultures from the gums in cases of parodontal disease.

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[1] *Medical World*, December 4, 1913, 688.

[2] The use of the term 'elastic' in this quotation must not be taken too literally; there is no elastic tissue in the periodontal membrane.

[3] *Lancet*, 1935, 2, 869.

[4] *Proc. Roy. Soc. Med.*, 1936, xxix, 1552.

In parodontal disease the area from which absorption can take place is often extensive.

It has been estimated[1] that if all the teeth are affected by advanced parodontal disease, the additional surface area brought by this means under external influences is just over twenty square inches. Such an extreme condition is seldom seen, but it is not uncommon for a third of the surface area of the teeth to be involved in the disease. It must not be thought, however, that the whole of this surface is capable of absorbing toxins; this extra surface (the lining of 'pockets') becomes epithelialised over as it is formed, and there are only occasional breaches of this surface, though one usually finds that most of this epithelial surface is infiltrated with inflammatory cells.

(E) INCIDENCE, CLINICAL APPEARANCES, MORBID ANATOMY, AND PATHOLOGY IN ANIMALS.

(1) Captive and Domesticated Animals.

Considerable light has been thrown on the pathology of parodontal disease by a study of the disease in animals.

In **animals** that have been the subjects of this disease exactly similar changes are to be found in the bones as in man, but the teeth show greater deposits of calculus owing to the absence of the system of artificial cleaning adopted by human beings, and specimens often show the spaces between the teeth choked up with food debris.

The following specimens show the different stages of the disease in animals :—

An early stage is seen in fig. 657, a Bennett's wallaby (*Macropus bennetti*). In the maxilla the septa between the anterior

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[1] 'Pyorrhœa Alveolaris.' D. A. Crow.

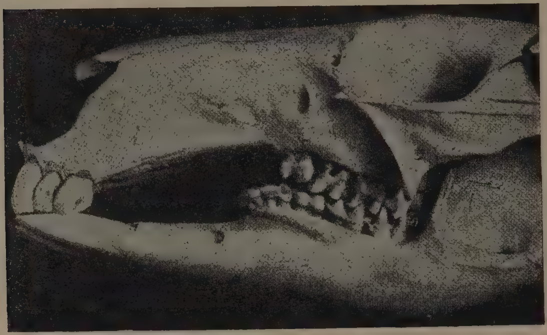

Fig. 657.—*Macropus bennetti* (Bennett's wallaby) showing an early stage of parodontal disease. The loss of bone is more marked in the maxilla than in the mandible.

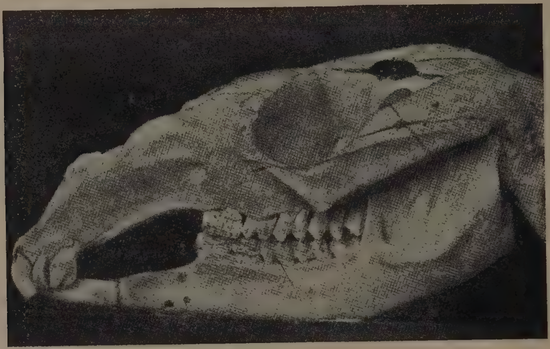

Fig. 658.—*Petrogale penicillata* (Brush-tailed kangaroo). In this specimen the disease is more advanced than in the specimen fig. 657.

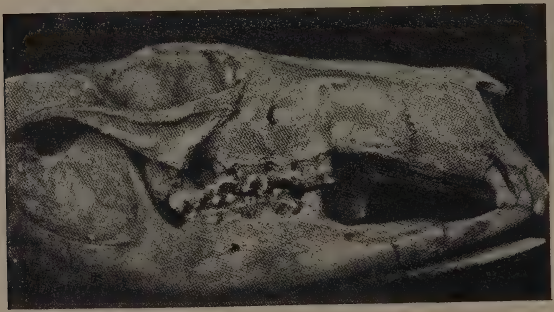

Fig. 659.—Macropus—species unnamed. The disease is well advanced and is more marked in the maxilla than in the mandible.

teeth have been partly destroyed and in the region of the first molars the bone shows signs of rarefying osteitis. A more advanced stage is shown in the skull of a brush-tailed kangaroo (*Petrogale penicillata*), fig. 658. Here the disease is more widely distributed.

The next specimen (fig. 659) illustrates a further stage. The bone has been extensively destroyed in the neighbourhood of the maxillary right molars, and what remains shows signs of rarefying osteitis. The disease is more advanced in the maxilla than in the mandible. In the left half of the skull and mandible (fig. 660) the disease has caused the loss of all the cheek teeth with the exception of one molar in the maxilla and one molar in the mandible.

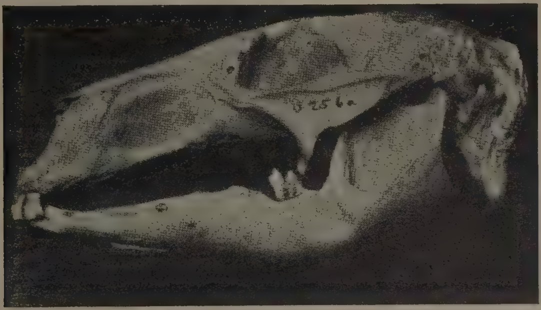

FIG. 660.—Macropus—species unnamed. The majority of the teeth have been lost from parodontal disease.

In carnivora a similar gradational series can be traced, the disease tending to spread more rapidly and extensively in the maxilla than in the mandible owing to the less dense character of the former bone (see figs. 661 and 662).

In herbivora the infection does not tend to spread far beyond the area affected. The localisation of the infection is particularly noticeable in the maxillæ of herbivora, and this is probably due to the fact that in the herbivora this bone is denser in character than in the carnivora. This point is shown in the skull and mandible of a Panolia deer (fig. 663).

Horses.—In the course of an examination of nearly 500 skulls of horses which had worked in London it was found that approximately one-third presented some degree of parodontal disease, ranging from a slight destruction of the gingival margin to the most aggravated form of the disease.

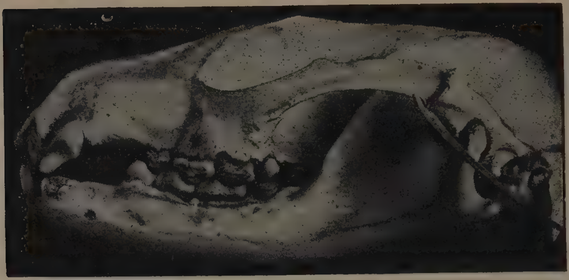

Fig. 661.—*Paradoxurus larvatus* (Chinese palm civet). The destruction of the bone is more marked in the maxilla than in the mandible.

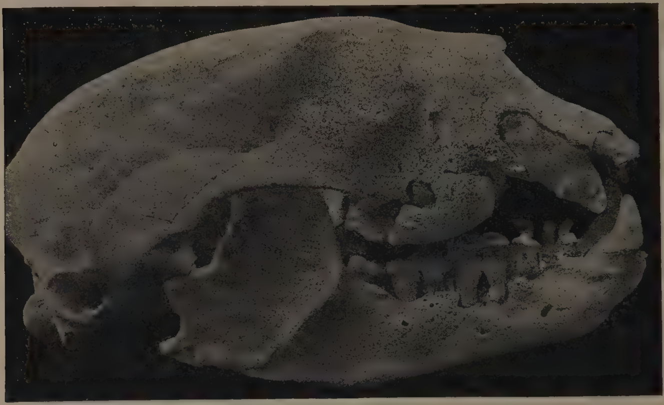

Fig 662.—*Tremarctos ornatus* (Spectacled Bear). R.C.S. Odonto. Section G, 143, 1. The specimen shows an advanced stage of parodontal disease on the right side. (From 'Variations and Diseases of the Teeth of Animals.' J.F.C.)

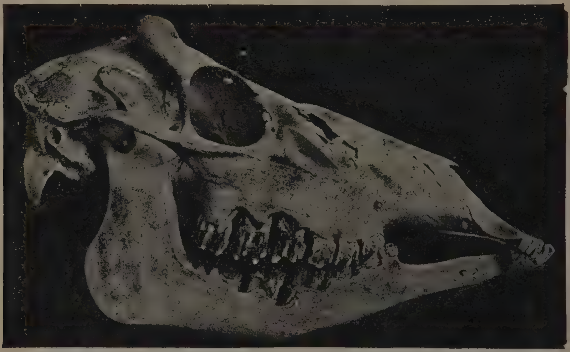

Fig. 663.—*Cervus eldii* (Panolia deer). The destruction of the bone is most marked in the region of the first molar.

The first sign of mischief is a slight injury to the gingival margin, resulting in the destruction of the muco-periosteum filling up the intervals between the teeth. These spaces are usually seen in the maxilla on the buccal side and in the mandible on the lingual side. In the spaces formed, food, etc., collects and undergoes fermentation, leading to the destruction of the adjacent tissues, so that in time a marked space is formed between the teeth into which fodder or other material may become firmly wedged (figs. 664 and 665). As the disease progresses the

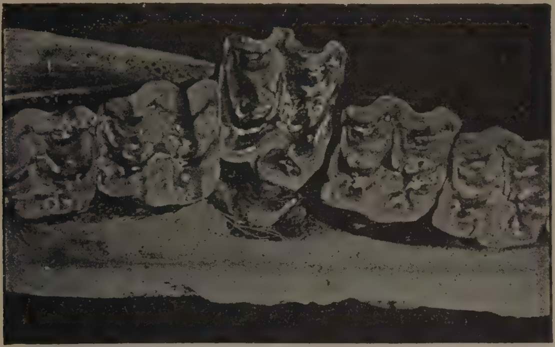

FIG. 664.—Portion of maxilla of horse, showing destruction of the bone around the teeth. The disease is most marked in the neighbourhood of the first molars. (From *Trans. Odonto. Soc.*)

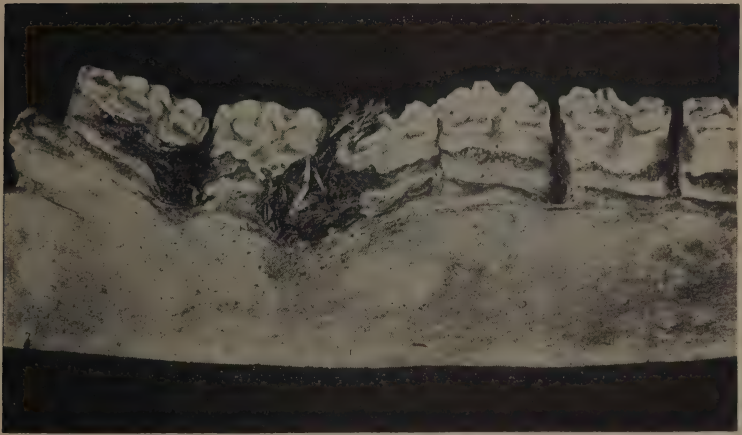

FIG. 665.—Portion of the mandible of a horse, showing food packed between the teeth. (From *Trans. Odonto. Soc.*)

parodontal tissues become more and more involved, leading to extensive destruction of the bone ; the teeth loosen, and the infection spreads in the maxilla to the sinus, setting up suppuration, and in the mandible to the body of the bone, causing an abscess.

If a horse in which the disease is advanced be examined, the muco-periosteum is seen to be inflamed and thickened (see fig. 666) and a profuse muco-purulent discharge will be noticed around the teeth, the breath being extremely offensive. The mandible is much more liable to attack than the maxilla, and, as far as one's observations go, more spaces are met with between the anterior premolars than between the molars, but the spaces formed between the premolars do not apparently set up the same amount of trouble as those found towards the back of the mouth. In the maxilla spaces seem to occur with the greater frequency towards the back of the series. As a result of the spaces the teeth become painful to pressure, and the function of mastication is interfered with, leading in many cases to marked irregular wear of the teeth. In some cases only one or two spaces are seen, in others huge gaps occur between nearly all the teeth.

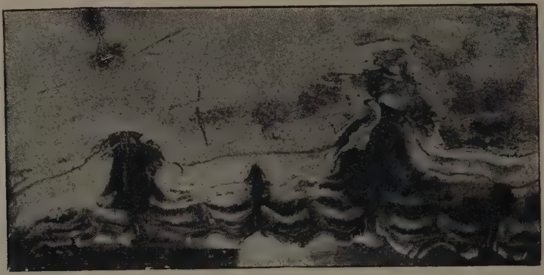

Fig. 666.—Portion of the mandible of a horse with the muco-periosteum in position. The effect of the food packing on the soft tissues is well shown. (From *Trans. Odonto. Soc.*)

The incisors may be attacked by parodontal disease. The effect on the incisors is in many respects not unlike that seen in the human subject ; the alveolar process disappears ; the teeth loosen, become forced out of place, and are finally lost.

The facts that the spaces start on the buccal sides of the maxillary teeth and on the lingual aspects of the mandibular teeth, and that the bone destruction is greatest in the region where the food is most liable to collect, would seem to suggest that food lodgment plays an important part in the causation of the disease.

In the horse the disease would seem to be mainly local in origin. The injury of the muco-periosteum is probably due to foreign bodies in the diet. It may arise from food, prepared in the form of chaff or grains of corn, becoming wedged in the 'gingival trough.' To the fermenting food in the spaces, infection is added from organisms present in, or introduced into, the mouth, and from them and the toxins they produce the tissue destruction proceeds. With the loss of the teeth a nidus for the accumulation of debris, etc., no longer exists, and healing occurs.

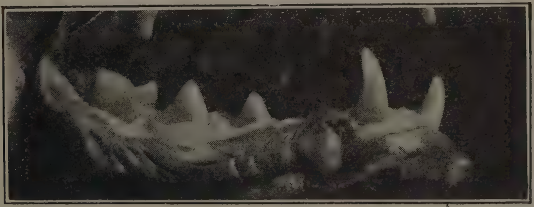

Fig. 667.—Normal gums in a cat.

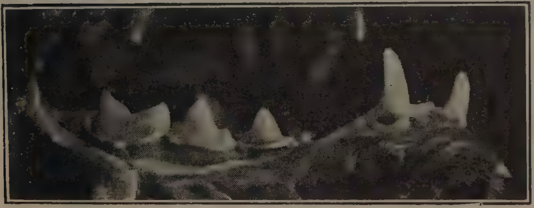

Fig. 668.—Marginal gingivitis in a cat.

Cats and Dogs.—Amongst domestic cats and dogs parodontal disease is extremely prevalent. H. Gray, a veterinary surgeon who has had considerable experience of the disease in these animals, is of the opinion that amongst cats the disease is most frequently seen in highly bred pet animals fed on soft food; the ordinary domestic cat fed on a meat diet keeping comparatively free from the disease. The explanation is that the soft diet clings about the teeth, stagnation areas are formed, and a marginal gingivitis results. This condition is well shown in fig. 668, and the appearance of normal gums in a cat for comparison in fig. 667.

Amongst dogs the pampered lap dogs, and the short-muzzled breeds, such as the pugs and bulldogs, are most subject to the disease, the non-functional teeth, *i.e.* the incisors, being specially prone to attack (figs. 669 and 670). Dogs living on a flesh diet, which gives plenty of exercise to the jaws, are invariably free from the disease.

Amongst cats and dogs, therefore, the disease is evidently closely connected with the soft pap diet which clings about the teeth, and ferments and injures the gingivæ.

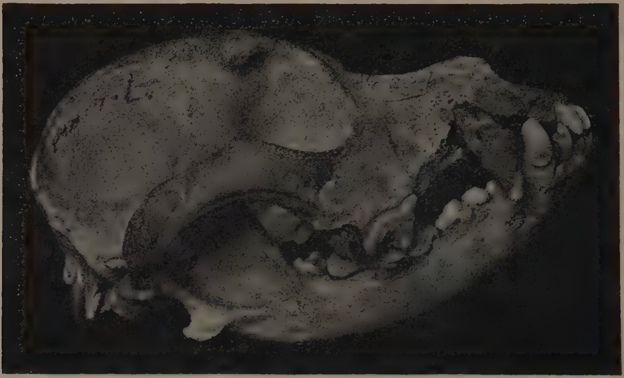

Fig. 669.—*Canis familiaris* (Domestic Dog). R.C.S. Odonto. Section G, 145, 7. This specimen shows an irregular distribution of parodontal disease. (From 'Variations and Diseases of the Teeth of Animals.' J.F.C.)

In animals kept in captivity parodontal disease is of frequent occurrence, and the Museum of the Royal College of Surgeons of England contains an excellent series showing the disease in a wide range of animals, *i.e.* herbivora, rodents, marsupials, monkeys, the small carnivora, etc.

In the skulls of captive animals affected by the disease it will be noticed that the bone destruction is usually most advanced around the areas where the greatest force of mastication is exerted and consequently where the soft tissues surrounding the teeth are most likely to be injured. The frequency of the disease in captive animals and the comparative immunity of animals in the wild state, taken in conjunction with the limitation of the disease to the masticating area, point to the disease being closely associated with the character of the food given to captive animals. This food is generally of a softer character, needing less rending and tearing than the food which the animals would obtain in the wild state; consequently, the teeth are less used and the natural cleansing operations are not performed. When captive wild animals are given food of a character similar to their natural food, as in the case of the large carnivores which are fed on a diet of fresh meat, they are almost entirely free from the disease.

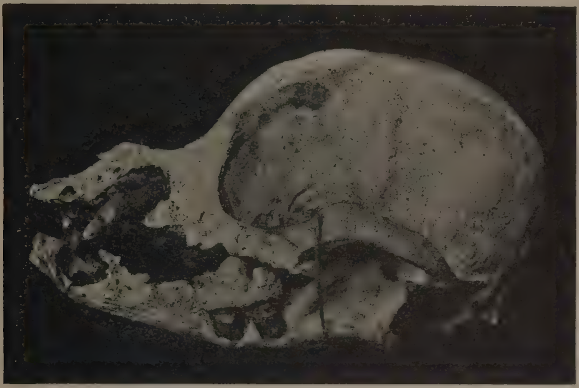

FIG. 670.—*Canis familiaris* (Domestic Dog). R.C.S. Odonto. Section G, 145, 8. An advanced stage of parodontal disease. (From 'Variations and Diseases of the Teeth of Animals.' J.F.C.)

(2) Wild Animals.

Monkeys living in the wild state are not immune to destruction of the alveolar process.

In the **New World Group** Brachyteles shows a considerable tendency to injury of the alveolar bone. In seven out of twelve adult specimens collected from the same district there was lipping or cupping of the alveolar process in varying degrees, and in one specimen several of the teeth had been lost from disease. In Pithecia the variety Satanus seems susceptible, for in six adult specimens from the same district three were affected. The genera Mycetes, Chrysothrix, Hapale, and Midas are liable to the disease, but in Uacaria, Callithrix, and Douroucollis it is apparently uncommon.

In the **Old World Group** parodontal trouble is comparatively common in Semnopithecus. The teeth of these animals are bulbous at the necks, and it is possible that the food is easily caught in the interdental spaces. The incidence of the disease differs in the various species; for example, it is uncommon in *S. obscuras*, but is fairly frequent in *S. cristatus pallata* and *S. entellus anchises*. In Pyrrhus, a variety not particularly liable to the disease, a series of ten specimens from Java all showed recession of the alveolar process, and in four cases definite pockets had formed.

In wild animals in their natural state generalised destruction of the bone is extremely rare, but localised areas of destruction are by no means rare. The following table[1] shows the number of pockets seen in different genera of monkeys :—

Genus.	No. of Specimens.	No. of Specimens with Pockets.	Percentage of Specimens with Pockets.
Colobus	1205	49	4·1
Semnopithecus ...	289	21	7·3
Trachypithecus ...	385	30	7·8
Presbytis	311	33	10·6
Cercopithecus	1473	22	1·5
Macaca	588	34	5·8
Papio	288	0	0·0
Cebus	538	6	1·1
Ateles	186	4	2·2
Alouatta	781	20	2·6
Hapale	164	20	12·2

In Macacus destruction of the alveolar process is uncommon, but in fifteen specimens of *M. fascicularis* from Java six showed considerable localised destruction of the bone, and in nine specimens of *M. fuscatus* from Takushima five showed definite pockets.

Of the genus Cercopithecus two hundred specimens were examined and only nine showed much bone destruction, and these were from British East Africa and the Congo region.

The fact that in certain varieties of monkeys the disease is found in some localities and not in others suggests that the presence or absence of the disease is to be attributed to the character of the food available in the particular district. In this connection it is of interest to record that specimens of the Golden

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[1] 'Variations and Diseases of the Teeth of Animals,' 1936, 684. J.F.C.

Mole (*Chrysochloris aurea*) from the Stellenbosch district in Africa all show early loss of the teeth. Dr. Broom [1] states that this condition is not common in other districts where the Golden Mole is found.

Though rare, this disease has also been found in wild specimens of ungulates and marsupials, but is practically unknown in wild carnivores and rodents. Figs. 671 to 674 illustrate progressive cases of the disease occurring in wild primates.

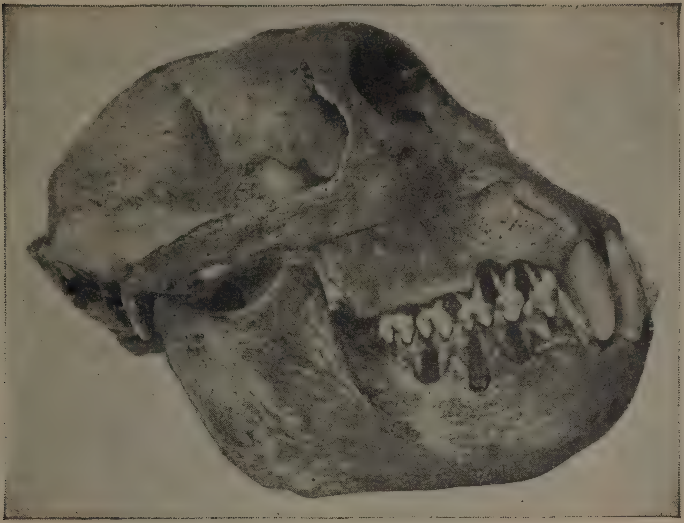

FIG. 671.—*Colobus badius tephrosceles* (Uganda Red Colobus). B.M. 30.8.1.3. This specimen shows pockets between the teeth. Food is present in the pocket between the mandibular first and second molars. (From 'Variations and Diseases of the Teeth of Animals.' J.F.C.)

The study of the disease in animals would seem to show that it is purely local in origin, that is, it is due to the lodgment of food; the lodged food undergoes fermentation and putrefaction and the tissues are damaged. This view is supported by the condition of the gums and teeth of cattle in parts of America, where, owing to the penetration of the tissues by the barbed crowns of *Hordeum jubatum*,[2] destruction of the teeth sockets occurs and the teeth are

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[1] Private communication.

[2] 'Dental Surgery,' Tomes and Nowell, 648.

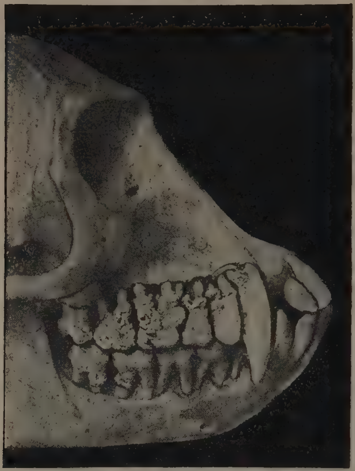

FIG. 672.—*Cercopithecus.* R.C.S. Odonto. Section G. 167.2. A case of rapid destruction of the alveolar process. (From 'Variations and Diseases of the Teeth of Animals.' J.F.C.)

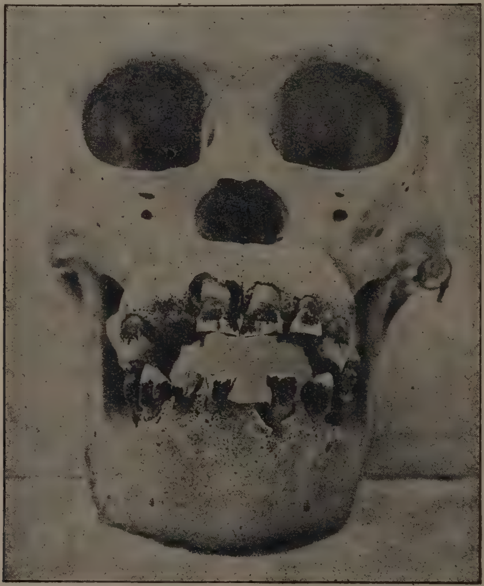

FIG. 673.—*Anthropopithecus troglodytes* (Chimpanzee). The Powell-Cotton Museum M. 475. Several of the teeth have been lost; the sockets of some of the teeth have been destroyed. (From 'Variations and Diseases of the Teeth of Animals.' J.F.C.)

lost. G. Thomson[1] states that parodontal disease is common in horses running almost wild in countries liable to prolonged drought. The animals under these conditions dig up roots, and in chewing the grit and sand injure the gums and start parodontal disease.

E. S. Talbot[2] has shown that in dogs the earliest manifestation is in the gingival margin, and that with the progress of the disease both the periodontal membrane and the bone become involved.

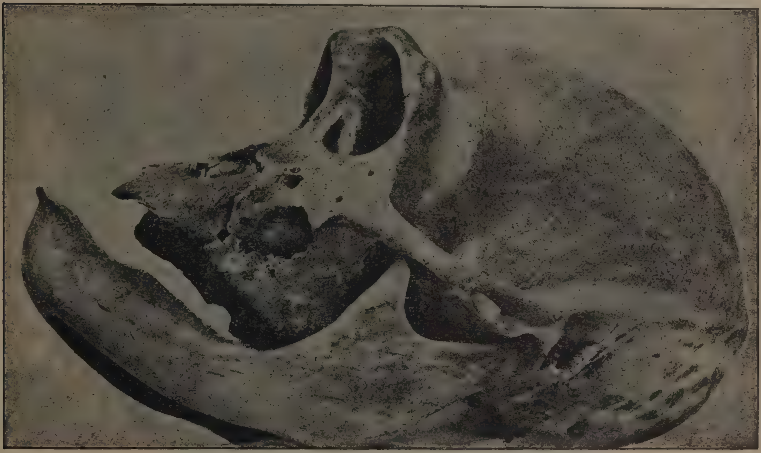

FIG. 674. — *Anthropopithecus troglodytes* (Chimpanzee). The Powell-Cotton Museum M. 165. A specimen showing loss of the teeth. (From 'Variations and Diseases of the Teeth of Animals.' J.F.C.)

The absorption of the bone is at times rapid and takes place by (*a*) lacunar absorption ; (*b*) the formation of perforating canals ; and (*c*) halisteresis (disappearance after prior absorption of lime salts).

(F) BACTERIOLOGY.

The Relationship of Micro-organisms to the Disease.[3]

It is well recognised that the bacteriology of parodontal disease is in a most unsatisfactory state, and that there is as yet no proved relationship between the micro-organisms found in the disease and the disease itself. All that can be said at present is that no specific micro-organism or group of micro-organisms can be held responsible for the condition we speak of as pyorrhœa.

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[1] *Trans. Odonto. Soc.*, xxxviii, 76.

[2] 'Interstitial Gingivitis.'

[3] For the details of the bacteriology of parodontal disease we are indebted to Arthur Bulleid.

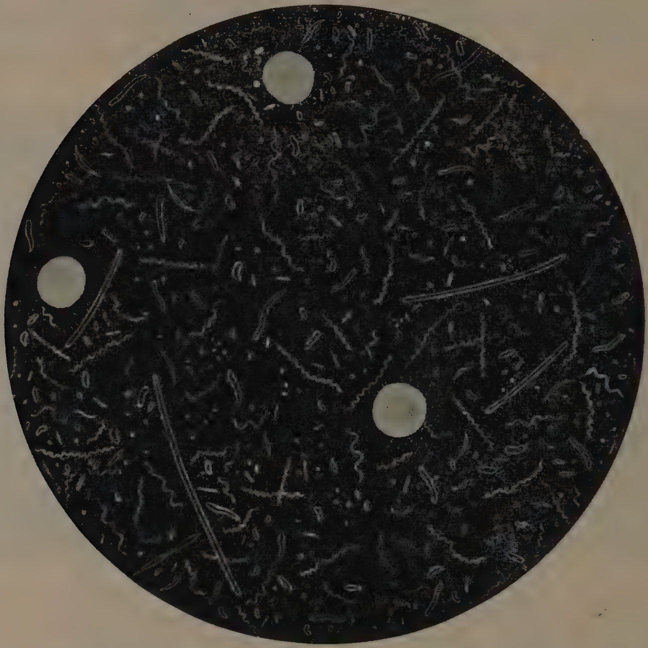

Fig. 675.—Diagram of organisms seen in coverslip preparation by dark ground illumination from a case of parodontal disease, showing: *Treponema macrodentium* and *microdentium*. Vibrios and spirillary forms. Bacilli in great variety. Filamentous forms. Cocci of various sizes, the grouping of which cannot be determined without cultivation methods. The large round bodies in the preparation are blood cells. Discharge from the same pocket examined by transmitted light revealed *Amœba buccalis*. (W. J. Penfold.)

It may be found that the actual micro-organisms isolated are in fact only secondary invaders.

If a smear is made from the exudate expressed from one of the deeper pockets and examined either under dark ground illumination or in a preparation stained by one of the simple watery aniline dyes such as methylene blue, it will reveal a vast assortment of micro-organisms (fig. 675). Examples of the following groups are usually found: (A) Bacteria; (B) Protozoa; (C) Yeasts.

(A) BACTERIA.

Cocci.—Streptococci; Pneumococci; Staphylococci; *M. tetragenus* (all Gram positive); *M. catarrhalis* and allied Gram negative diplococci.

Bacilli.—*B. influenzæ*; B. of Friedlander; some indifferent Gram positive and Gram negative bacilli; and *B. fusiformis* (anærobe).

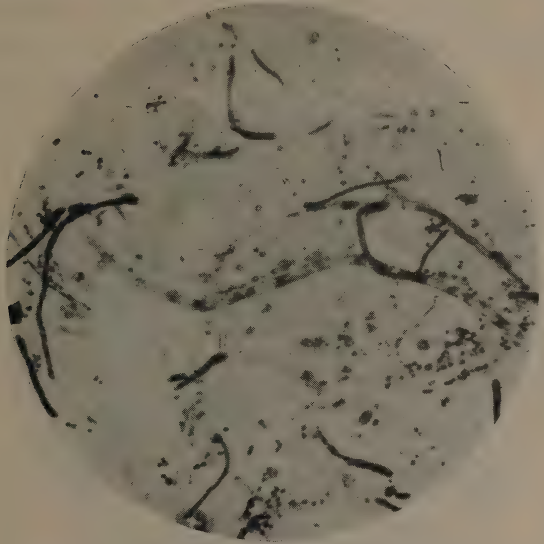

FIG. 676.—*Leptothrix buccalis.* (Arthur Bulleid.)

Spirochætes.—*S. Vincenti*; *S. refringens*; *T. macrodentium*; *T. microdentium*; and *T. mucosum* (Noguchi).

Leptothricæ.—*Leptothrix buccalis* and allied thread organisms (fig. 676).

(B) PROTOZOA.

Amœbæ.—*Amœba gingivalis* and *Amœba limax* (Glynn) (fig. 677).

(C) SACCHAROMYCES.

Yeast forms are occasionally found. It must be noted that it is practically impossible to identify the actual micro-organisms by direct smear alone, and cultural methods must be undertaken in order to differentiate the various organisms found. Even when careful ærobic and anærobic cultivations are carried out quite a large proportion of the immense flora fail to grow.

CULTURAL FINDINGS.

If cultures are made from the exudate expressed from the pockets in a case of parodontal disease, the following micro-organisms are at times isolated.

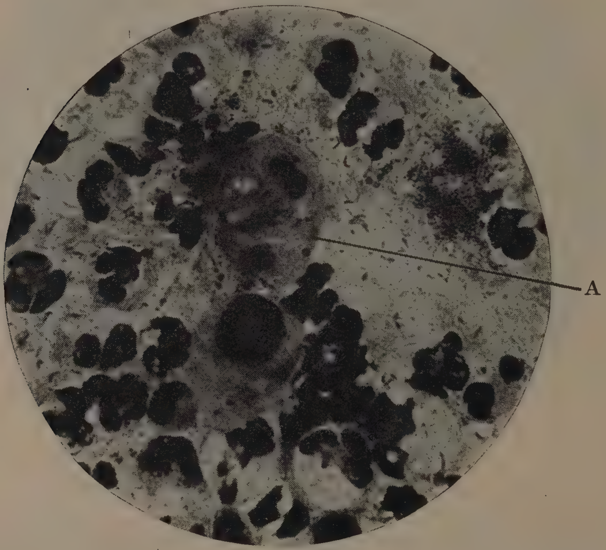

FIG. 677.—Pyorrhœic pus.
A. *Amœba buccalis.* (Arthur Bulleid.)

Ærobic Cultivation.—Streptococci (usually the *viridans* group); Pneumococci; *M. catarrhalis* and allied Gram negative diplococci; *M. tetragenus*; *B. influenzæ*; B. of Friedlander. Staphylococci, both the *albus* and *aureus* are found, but they are relatively uncommon as compared to the Gram negative cocci.

Anærobic Cultivation.—The same micro-organisms as mentioned above and occasionally *B. fusiformis*.

It will be noted that the micro-organisms most frequently isolated in cases of parodontal disease are those which are frequently found in suppurative processes in other parts of the body and no specific causal micro-organism has as yet been isolated.

Under these circumstances the reaction found in the tissues involved is of more importance than the organisms found in the pockets.

Résumé.

A general survey of the morbid anatomy, pathology, and clinical appearances leads to the conclusion that the initial lesion in man is injury to the gingival margin, which results in the formation of a stagnation area. In this area material collects from which toxins are formed, and the tissue changes which follow are identical with those produced in the lower animals. In the area immediately beyond the irritated surface—for as such it must be regarded—the tissues react and form a protective barrier, and the progress of the disease is governed by the efficiency of this barrier. The reaction of the tissues depends upon :—

(*a*) Their inherent vitality.

(*b*) The degree of functional activity—the greater the flow of lymph through the area the greater will be the reaction.

(*c*) The severity of the infection.

In many cases, more especially in young adults, the natural defence of the tissues suffices to prevent the undue absorption of toxins or organisms via the bone. But the discharge from the mouth is swallowed with the saliva and consequently the gastric mucous membrane is continually being damaged. Sooner or later the defence of the tissues at this part breaks down and eventually the whole intestinal tract becomes involved.

The tissues of many patients seem to be incapable of raising an effective barrier even in mild cases. In cases where the natural defences of the tissues around the teeth are inadequate some of the toxins produced pass directly into the general circulation and some escape into the mouth (open sepsis) and are swallowed.

(G) ETIOLOGY.

Considerable diversity of opinion exists as to the cause of parodontal disease.

In animals it has been shown that the lesion is caused by injury from the food. When cats and dogs are fed on a diet of fresh flesh, their natural food, they are invariably free from the disease, but as soon as soft pap food is substituted the animals become a prey to the disease. In the horse the disease can clearly be traced to injury from the food, the particular form in which the food is given being responsible for the injury. Under natural conditions horses feed upon grass which is torn up in long strands and in a pliable state, and is masticated and disposed of without injury to the soft tissues. Under 'civilised' conditions horses frequently cannot be put out to grass and the food is often given in the form of hay and straw (chaff) cut up into short lengths. Chaff in short stiff lengths is very likely to cause injury to the muco-periosteum through being pressed into spaces between the teeth. This view of the causation of the disease in horses is supported by the fact that the disease is prevalent wherever the conditions are such that fragments of thorn become mixed with the food. Similarly, as previously mentioned, cattle in parts of America are subject to the disease owing to the penetration of the tissues by the barbed crowns of *Hordeum jubatum*. In the horse the disease is clearly to be attributed in a very large measure to the physical character of the food.

In **man,** too, *the evidence points to the disease being started by injury of the gingival margin from food and other debris, and/or the local action of micro-organisms and their toxins as seen in the marginal gingivitis of mouth-breathers.*

The prevalence of the disease is probably due to the character of the diet of the present day. Much of our food is now prepared in such a manner that it readily accumulates around the teeth and is of a character which easily undergoes fermentation. The food thus prepared requires but little mastication, with the result that the tissues in and around the teeth are deprived of an adequate blood supply and are thus rendered less resistant to attack.[1] As soon as the disease has started, the formation of abnormal pockets favours the accumulation of debris, thus increasing the basis of infection and so accelerating the action of the toxins.

Attention has been drawn to the intimate **relationship between this disease and 'mouth-breathing,'** and the increase in the number of individuals who are mouth-breathers has, undoubtedly, added to the incidence of the disease.

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[1] Evidence in favour of this is shown in a series of skulls of Londoners of the sixteenth and seventeenth centuries. The disease in the region of the molars is extremely common in the skulls, but the disease is chronic in character and

The view here expounded that stagnation of food is the primary cause is supported by an observation made by S. Colyer [1] on the teeth of the Barotze, a tribe living in North-West Rhodesia. Their diet is mainly of a carbohydrate character to which milk, fish, and meat are added as savouries. They do not clean their teeth and suffer markedly from the disease. On the other hand, the native police of the district, who are drawn from Zulu, Central African, and Arab strains, although eating the same type of foods, have very little disease. The police clean their teeth with sticks twice a day and always after the evening meal.

It is believed by many that conditions of *sub-scurvy*, occasionally even approaching scurvy, may have considerable etiological importance especially in initiating this disease in children, and inquiry should always be made to exclude this possibility as a factor, or, if it exists, to treat it by the administration of ascorbic acid.

(H) TREATMENT.

In the section on the morbid anatomy and pathology of the disease, it was pointed out that the formation around the teeth of 'pockets,' which inevitably become stagnation areas, is the outstanding feature of the disease. Until these 'pockets,' or stagnation areas, have been eradicated, it does not seem reasonable to claim that a cure has been effected, and as it is almost impracticable to eradicate them actual cures are rare, though well-directed treatment may keep the disease in check. Reference has also been made to the important rôle which mouth-breathing plays in the promotion of the disease. It has also been shown that the progress and severity of the disease are greatly influenced by the degree of resistance offered by the tissues.

Treatment, therefore, should be directed towards promoting efficient drainage, raising the resistance of the tissues and ensuring proper nasal breathing and functioning of the teeth.

With regard to the breathing, we are firmly convinced that in all cases of 'mouth-breathing,' even if the patient is addicted to

the bone shows marked reaction to injury. In the majority of these skulls the function of mastication was efficiently carried out, as is shown by the attrition of the teeth.

[1] *Dental Record*, October 1920.

this habit for a few hours a day only, there is very little hope of checking the disease; moreover, in such cases the general symptoms seem more pronounced. It is essential, therefore, to ensure proper nasal breathing. Where the patient keeps the lips apart the nasal cavity must be examined and any obstruction removed. Some patients persist in the habit of 'mouth-breathing' after a free nasal passage has been obtained. The habit can, however, often be overcome by the use of an oral screen (see p. 253).

The **therapeutic measures to be adopted** may be divided into (1) Local and (2) General.

(1) Local Treatment.

Local treatment consists in the promotion of asepsis as far as practicable by securing efficient drainage. Efficient drainage relieves the tissues of the constant injury arising from the toxic matter around the teeth and the tissues react more readily. It may be confidently affirmed that the more thoroughly the pockets are deleted, the greater will become the prospects of staying the progress of the disease. In theory efficient drainage would seem an easy matter to establish; in practice, however, such is not the case. The local remedies employed should be directed towards :—

(*a*) Freeing the teeth from salivary and serumal calculus and other debris.
(*b*) Reduction of pockets and cleansing of pockets and interproximal spaces.
(*c*) Surgical deletion of pockets (gingivectomy).
(*d*) Massage of the gums.

(*a*) The removal of calculus removes a source of irritation and infection, allows the pockets to drain more freely, and facilitates irrigation. This must be done thoroughly and special care taken to remove any calculus in the interstitial spaces. Where there is much gingivitis it is advisable to delay the removal until the gingivitis has been reduced by appropriate treatment, and the general resistance raised by improving the general health. The operation of removal of calculus can then be carried out more efficiently and with less laceration of the tissues, which are now less in bulk on account of decreased œdema.

(*b*) In considering the treatment of pockets, it must be remembered that they are deepest at the most inaccessible parts, *i.e.* in the interspaces. The regular use of astringents will often produce good results. Tannic acid in the form of a powder may be used and should be rubbed into the gums for two minutes once a day, the treatment being continued for at least two months. If at the end of this period the gum margins have shrunk, the application may be reduced from once a day to twice a week. As tannic acid is liable to leave an indelible stain on linen, patients using it should be warned against wiping their hands on linen.

Regular cleansing of the pockets and interproximal spaces by the patient is an essential part of the local treatment. First, the spaces between the teeth must be freed of food debris by means of wooden toothpicks, 'floss silk' or worsted; the gums should then be well squeezed between the thumb and first finger with a downward pressure on the upper teeth and an upward pressure on the lower teeth. Irrigation is here of value. If the irrigation can be easily carried out, a free flushing with hydrogen peroxide (two volumes) is very effective. The most efficient method is to use a hypodermic syringe. The needle of the syringe is passed well into the pocket and gentle pressure exerted. The use of force is to be deprecated as it is essential that any granulation tissue that may be present should not be broken down. But as a hypodermic syringe cannot be readily manipulated by patients it is generally necessary to try an easier, if less efficient, means of irrigation.

A good plan is to have the pockets and interspaces wiped out with a wisp of cotton-wool dipped in hydrogen peroxide, the cotton-wool being wound round a fine broach. It is necessary to give patients very precise instructions. The broach should be of the type ordinarily used for root canal dressings. The method of winding the wool on the broach should be demonstrated. The following plan is suggested: Spread a wisp of cotton-wool along the palmar surface of the terminal phalanx of the first finger of the left hand; place the broach on the centre of the wool; fold the cotton-wool over the broach; bring the thumb on to the broach and then run the thumb with the broach up the palmar aspect of the first finger; the fragment of wool over the point should then be turned down so as to prevent the point sticking through.

Such detailed instructions may seem hardly necessary, but experience teaches that the success of this method of treatment depends in a large measure on the skill of the patient in twisting the wool on to the broach.

The patient should be shown exactly where and how to apply the peroxide of hydrogen and instructed to pay special attention to the irrigation of the spaces between the posterior teeth. The hydrogen peroxide to be used should be poured into a small receptacle, and any left unused should be thrown away and not returned to the bottle. The pockets should be cleansed at least once a day, the best time being shortly before retiring for the night. If the patient will only carry out this method of cleansing the spaces thoroughly and regularly, a marked improvement may be confidently anticipated.

Hydrogen peroxide is an antiseptic, and its beneficent action is partly due to this property, but it has great additional value; by its action on organic tissue it gives off oxygen, which may act as a local stimulant, and the profuse frothing which it produces helps to dislodge particles of food, dead epithelial cells, micro-organisms and other debris which cannot readily be dislodged by other means owing to difficulty of access. The drug in 2-volume strength may be used as a mouth wash, but if it is used in too strong concentrations it is apt to destroy tissue without remaining long enough to sterilise it, and so may provide a further nidus for bacterial growth.

Copper sulphate, used in the powdered form, is also of value as an antiseptic and astringent in helping to reduce the depth of pockets, but is objected to by many on account of its unpleasant taste, slow solution, and the salivation it is apt to produce, or even vomiting if swallowed.

Many other drugs have also been used for local treatment, of which, perhaps, tincture of iodine, zinc chloride, and quinine sulphate are among the most useful.

The above treatment will often be found sufficient for cases where the depth of the pockets does not exceed 2 mm. on either lingual, or buccal and labial aspects; but more difficulty will be experienced in dealing with the interdental spaces. These may be packed firmly enough to cause slight local blanching with cotton-wool wisps impregnated with a mixture of zinc oxide and eugenol for a few days, the packing being so as to displace the œdematous interdental papillæ lingually and buccally. When the packing is removed any remaining calculus may then be removed, and any protuberant tags of granulations or interdental papillæ may then be removed by the electro-cautery or a chemical caustic.

(*c*) If, however, the pockets are deeper, after the preliminary treatment to get rid of inflammatory œdema and render the field of operation as clean as possible, the surgical removal of the redundant gum may be necessary, as *the entire deletion of the pockets is the essentially important item in the successful treatment of this disease.* This operation is known as **gingivectomy**, and may, if the field of operation is clean, be performed under local anæsthesia.

As a preliminary it is desirable and essential to register the depths of the pockets accurately ; for this purpose an ingenious method is that described by J. C. P. Besford, who writes : ‘ dressing tweezers may be so modified that the tip of one blade carries a sharp point set at right angles to the other blade which is inserted in the pocket. On closing the tweezers the gingiva is pierced at a spot corresponding with the epithelial attachment to the tooth and a bleeding point results.’ A series of such small punctures is made and then, with suitably shaped small scalpels or knives, an incision is made connecting all the punctures, and so dividing the gum as to leave it sloping away at a natural inclination. Any granulations or excess of papilla in the interdental space are then scraped or cut away. Packing of the interdental spaces should then be carried out with cotton-wool impregnated with a mixture of zinc oxide and eugenol as already described, and on its removal a week later the epithelialisation of the whole of the incised surfaces soon becomes complete.

Two-volume peroxide is now invaluable to help keep these spaces free of food debris.

The use of wooden points (toothpicks) is invaluable at this stage, and indeed whenever interdental tissue has been removed or lost so as to leave an interdental space, as both gross and fine food particles collect in these spaces at every meal. E. W. Fish strongly advocates the consistent massaging of the interdental spaces and gum margins by means of the wood points, with the view of so thickening the keratinised layer of epithelium over them, just as thickened callosities may occur on the hands when subjected to manual work ; it is uncertain if such can really occur on a mucous membrane, but rather that the return to normality may be brought about by the improved drainage caused by keeping the spaces free of debris, so that there is nothing left to injure or destroy such normally keratinised surface as is there.

(*d*) For the same reason consistent massage of the gums with the fingers, with a tooth-brush, and vigorous masticatory efforts on such foods as require it are valuable and will assist in improving the condition of the gingivæ.

Before leaving the question of local treatment, reference must be made to ionic medication in the treatment of parodontal disease. N. G. Bennett adopts the following technique in treatment: The teeth are freed of calculus and the mouth rendered as healthy as possible. It is not necessary—in many cases it is not possible—to remove all calculus as a preliminary to treatment; the process can be more completely accomplished during the course of treatment; indeed, small areas of marginal gingivitis which do not respond will often be found to be covering a small deposit of undiscovered calculus. A 5 per cent. aqueous solution of zinc chloride is generally employed, but salts of copper—such as the sulphate—or of silver, may be used; the electrode may be of platinum, or of the base metal of which the salt is being applied. A few contiguous teeth should be isolated and kept moderately dry by means of napkins, wool rolls, or bibulous paper. Absolute dryness is not necessary, but too much moisture will not only dilute the drug unduly, but also allow the current to pass superficially instead of through the tissues. The current having been turned on and the rheostat control advanced a short distance, the positive electrode, previously wrapped round with a small quantity of cotton-wool, should be dipped in the solution and applied to the gum at least a quarter of an inch from the neck of the tooth; it should then be slid carefully over the gum until the neck of the tooth is reached and passed down into the pocket round the tooth. The most important parts are the approximal pockets. It is usually convenient to treat about half a dozen teeth at a time, dealing first with the labial or buccal aspects and the approximal pockets, and completing on the lingual aspects. The amount of current registered with any given voltage varies inversely with the resistance of the body. This resistance itself varies with different patients and with the same person at different times. The amount of current that can be tolerated is usually from one to five milliamperes. It will usually be found that a current of one milliampere is not felt at all; more than five cannot usually be borne, except perhaps in the molar region. A current of three milliamperes is usually sufficient for therapeutic effect, and tolerable to the patient.

A current of low electromotive force having been tried at the outset, it can be gradually raised by moving the rheostat control until sufficient is obtained. It should be remembered that most pain will be felt when the needle is first applied (the 'make' of the current), less when it is broken, and least of all during the passage of the current, so long as the electrode is stationary. That is why the needle should, every time it has been dipped in the solution, be first applied to the gums and then moved to the tooth. If a large drop of liquid is hanging on the needle a considerable amount of pain may be felt at the first contact. Care should be taken to move the needle smoothly from one tooth to the next without making contact or even altering the area of contact more than necessary, because the current is felt more severely when passed through a small area than a large. When the teeth are very sensitive it is sometimes necessary to apply the needle first and then gradually turn the current on from zero; in this way pain may be minimised.

By taking groups of teeth in succession the whole mouth may be treated in about three-quarters of an hour, but it is often desirable to spend the greater part of the time over the most seriously affected teeth, three or four minutes being devoted to a single tooth. The only visible sign noticeable is a slight whitening of the edge of the gum, and a kind of white deposit spreading from the edge. It is only very rarely that any sensation is felt in the hand of the patient holding the negative electrode. It is desirable, however, that rings should be removed.

The application should be repeated every second or third day for a fortnight, then less frequently until about ten or a dozen applications extending over five or six weeks have been made. In this period it will be possible to judge results, and if considerable improvement cannot be obtained in that time or even less, it is not likely to be gained at all.

The action of ionisation is probably to stimulate the soft tissues around the teeth and sterilise the contents of the pockets, the drug exercising an astringent action. It is questionable whether it possesses advantages over the local treatment recommended on p. 640, but there is little doubt that deeper penetration of the tissues is obtained than by any kind of mere application of antiseptics. It should be understood that in cases at all advanced, or in which pockets remain after treatment, no cure can be expected; and that in all cases subsequent care on the part of the patient is essential. One constantly hears that the patient has undergone ionic treatment and is cured, but on inquiry no regular treatment is being carried out. In these patients the 'pockets' around the teeth still exist, and if not regularly cleansed the disease must progress.

(2) General Treatment.

General treatment, directed towards the removal of systemic intoxications, and a general improvement in health. The nearer the health approaches to the normal standard the greater becomes the resistance of the tissues and their power to react. An impairment of the general health implies a lowering of the resistance of the tissues and of their power to react, and it is very important therefore that steps should be taken to improve the general health as far as possible. But this part of the treatment belongs to the province of the general medical attendant.

Vaccine therapy has been freely used during recent years. A vaccine is a killed suspension of bacteria; on inoculation into the subcutaneous tissues the body produces antibodies which have a specific affinity for the particular organism present in the vaccine. The patient's serum may be examined with the view of obtaining evidence of the presence of antibodies to the organisms isolated.

The presence of antibodies may be shown by a number of different reactions. It should be borne in mind that agglutination, precipitation and complement fixation are different manifestations of an antigen-antibody reaction, the particular type of reaction being determined by the physical conditions of the experiment. In the past it was thought that each of these antibodies was distinct; but more recent work has shown their unity.

The object of vaccine therapy is to stimulate the production of antibodies which will enable the tissues to offer greater resistance to the infecting organism. Since antibodies are specific it is necessary to make a bacteriological examination to find out what types of bacteria are present in the focus. The method of procedure suggested by J. W. Eyre [1] is as follows : 'The patient is instructed to grasp the lip opposite the affected teeth with the forefinger and thumb of each hand and draw it away from the gums ; in some cases a small roll of absorbent cotton-wool is packed into the sulcus between the alveolar process and the lip. The gum margin is next wiped with a sterile swab of cotton-wool mounted on the end of a stick, and the gum itself dried with a second sterile swab. Then with another swab firm pressure is made on the gum over the root of the tooth. The first drop or two of pus that exudes is mopped up with a third sterile swab and the pressure continued, and the pus that next exudes is collected on still another sterile swab, or by means of a stout platinum needle. This pus is employed for the purpose of making coverslip films. Finally, more pus is expressed in a similar way and used to inoculate tubes of nutrient media.'

The most satisfactory method is to extract a tooth when this is practicable and take a culture from the space between the roots, if there is more than one, or from near the apex of the tooth ; the adjacent gum should be dried and painted with Liquor Iodi before the tooth is extracted or infection with organisms on the surface is very apt to occur during the operation. This does not by any means ensure sterility ; indeed, E. W. Fish claims that the actual cautery is the only certain means of attaining this end. Cultures of the various organisms present are then obtained *secundum artem*, and in any case a bacteriological report should be interpreted with caution.

The pathogenicity of the organisms isolated may be estimated by means of the bactericidal test, in which varying numbers of the bacterium under examination are added to constant volumes of defibrinated human blood ; saprophytic organisms are killed, whereas pathogens survive and multiply in the blood.

Only the pathogenic bacteria should be used for the preparation of the vaccine. The initial dose should be small (5 to 10 million organisms) ; subsequently, the dose may be doubled until 100 to 500 million organisms are given.

The injection of the vaccine must be carried out under strictly aseptic precautions and should be made subcutaneously.

As to the value of vaccine therapy in this disease there is extreme divergence of opinion. Vaccine therapy aims at assisting the tissues to defend themselves against the action of bacteria and their products. It is essential for success, therefore, that we should know the causative organism of the disease which has to be treated. The treatment by vaccine therapy, therefore, is not likely to be successful if stock vaccines are used. It may be urged in favour of vaccine treatment that the infection in the pockets is causing local injury, and that vaccines will check the injury by raising the resistance of the tissues in the neighbourhood of the tooth. The difficulty here lies in the fact that the infection is invariably mixed, and the vaccine therapist, if he desires to effect a thorough cure, must needs prepare a vaccine of all the pathogenic organisms found. If this is not possible it is the practice to use a vaccine of the predominant organism, or perhaps of two of them, and, under these conditions, the treatment is necessarily incomplete. But, granting that vaccine treatment is occasionally successful, the pockets still remain, and no amount of vaccine treatment will remove them.

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), iii, 34.

Apart, however, from any consideration which may be regarded as more or less theoretical, the question arises whether vaccines lead to good results in the treatment of the disease. One has had personal experience of many cases of parodontal disease which were treated with vaccines, and in no single instance was a cure effected and in only a few cases could one detect any improvement.

When vaccine treatment is carried out concurrently with local remedies and improvement results, it is extremely difficult adequately to assess the amount of improvement which should be ascribed to the local measures on the one hand and to the vaccines on the other. It is the general experience that the majority of cases treated with energetic local measures will show a rapid improvement.

In the treatment of the general condition arising from mouth infection vaccines may undoubtedly be extremely useful.

In papers advocating treatment by vaccines it is often claimed that a large percentage of the cases have been cured. Manifestly, much depends upon the exact meaning placed by the practitioner on the word ‘cured,’ and reference to this point has already been made (see p. 639).

The method of treatment to be adopted will depend upon the conditions present.

(*a*) Cases Favourable for Conservative Treatment.

A case may be regarded as favourable for conservative treatment if the following conditions are found :—

(*a*) The pockets around the teeth are shallow.

(*b*) The arch is well developed, and the function of mastication is efficiently performed.

(*c*) There are indications of recuperative powers on the part of the patient.

(*d*) The patient is a nose-breather.

If the pockets, which are the real cause of the trouble, cannot readily be removed, treatment resolves itself into a question of drainage. The more thoroughly the pockets are cleansed, the greater will be the chance of staying the progress of the disease. Calculus must be thoroughly removed, and the patient instructed to carry out the following daily routine :—

(*a*) Pass silk between the teeth to free the space of debris.

(*b*) Squeeze the gums so as to expel as much material as possible from the pockets.

(*c*) Brush the teeth and gums thoroughly with a stiff brush.

(*d*) Irrigate each space with peroxide of hydrogen, as suggested on p. 641.

(*e*) Twice a week apply to the pockets a 2 per cent. solution of iodine in alcohol.

These simple measures, if faithfully carried out, will suffice to arrest the disease in favourable cases. One has tried raising the resistance of the tissues by means of vaccines and Bier's method of congestion, but is not satisfied that these cases showed greater improvement than others where the treatment was limited to irrigation. Thorough cleanliness of the pockets is the keynote in treatment, and, if this is achieved, an arrest in the progress of the disease may be expected.

In cases where there are deep or even moderately deep pockets between the molars, it is often a good plan to isolate the second molars by the removal of the first and third molars.

(*b*) Cases not Favourable for Conservative Treatment.

Cases which are not favourable for conservative treatment are those which show well-marked signs of rarefying osteitis with general and local conditions indicating that the tissues possess little recuperative power; also all cases of persistent mouth-breathing. It will be convenient to group these cases under two separate headings, viz.:—

(i) Cases unassociated with apparent symptoms; and

(ii) Cases in which a causal relationship to other diseases has been established.

(i) *Cases unassociated with apparent symptoms.*

As regards cases falling under this heading, there are certain points which must be kept clearly in mind in endeavouring to arrive at the right line of treatment. Although efficient irrigation may check the discharge from the tissues, and some advantage may be gained in an attempt to raise the resistance of the tissues by the aid of auto- or hetero-inoculation, all such efforts will fail to stop the progress of the disease. In these circumstances the question arises whether it is justifiable to continue a course of treatment which, at the best, can do no more than slow down the progress of the disease. Our view is that such a course of treatment cannot be justified for two reasons: (1) Because a potential source of infection remains which may become active at any moment, and (2) because the destruction of the alveolar process is proceeding and thus increasing the difficulty of providing artificial dentures which will be efficient and comfortable.

(ii) *Cases in which a causal relationship to other diseases has been established.*

In this class of case it is absolutely necessary that the dental infection should be completely removed. After reviewing the cases which have come under notice during many years and carefully considering the results of different methods of treatment, one is satisfied that the removal of the affected teeth at the earliest opportunity is the right course to adopt, and that any other line of treatment will prove to be unsatisfactory. A typical example of this class of case is as follows: A young adult is suffering from rheumatoid arthritis and the mouth shows signs of this disease accompanied by rarefying osteitis in a marked degree. The case is one in which the potential source of trouble—that is, the pockets—cannot be removed by treatment. The choice here lies between retaining the teeth and running serious risk of an aggravated arthritic condition on the one hand, or, on the other hand, removing the teeth and ensuring useful joints. The latter course alone can be justified, as artificial dentures can be provided, but not new movable joints.

In carrying out extraction in these cases the following course should be adopted : The mouth is made as healthy as possible by thorough removal of calculus and irrigation of the pockets with hydrogen peroxide. The premolars and molars are next removed and the mouth is allowed to heal. Models of the mouth are then taken with the incisors in place and bites are obtained. The advantage of proceeding on these lines is that one avoids the difficulty which would be experienced in gauging the correct height of the bite after the removal of the front teeth. The anterior teeth are then removed and dentures inserted as soon as possible. The number of teeth which can safely be removed at one sitting depends largely upon the individual case. Where the ‘ power of repair ’ is at a low ebb the extractions must be carried out by easy stages, but where there is ample ‘ power of repair ’ the extractions may be carried out more expeditiously. Extensive extractions at one sitting should be avoided unless there are special reasons for adopting that course. It is said that trouble in the bone is likely to follow extensive extractions in these cases unless the resistance of the tissues is first raised by a course of vaccine treatment, but that is not one's experience. One is disposed to think that, where trouble in the bone occurs as a result of extractions, it is almost always due to the damage inflicted on the tissues by the operation. A rise in temperature seldom follows the extractions if the mouth is kept clean. Occasionally a rise in temperature does occur, as is well shown in fig. 678. The patient was suffering from a corneal ulcer, and the removal of each batch of teeth was followed by a rise in temperature.

In some cases it is found that the removal of a first batch of teeth causes very little general disturbance, whereas the removal of a further batch is followed by a considerable rise of temperature. As an example the following may be quoted : A man, about forty-eight years of age, had lost in five years two and a half stones in weight. There was marked parodontal disease. The $\left|\frac{4567}{4567}\right.$ were removed with no apparent septic sequelæ ; subsequently $\left.\underline{876}\,\right|$ were extracted, and within four hours there was a rigor with the temperature rising to 101° Fahr.

In cases where systemic trouble is traceable to the mouth it has been one's practice to retain the anterior teeth provided that the bone destruction is slight. The argument in favour of retaining the teeth in these cases is that the pockets around the anterior teeth are easily accessible and can be thoroughly irrigated by the patient.

Experience of these cases has taught one, however, that if the patients are mouth-breathers it is only a matter of time before extraction becomes imperative. It would therefore seem sounder treatment to remove all the teeth and thus make certain that the patient is freed from dental sepsis.

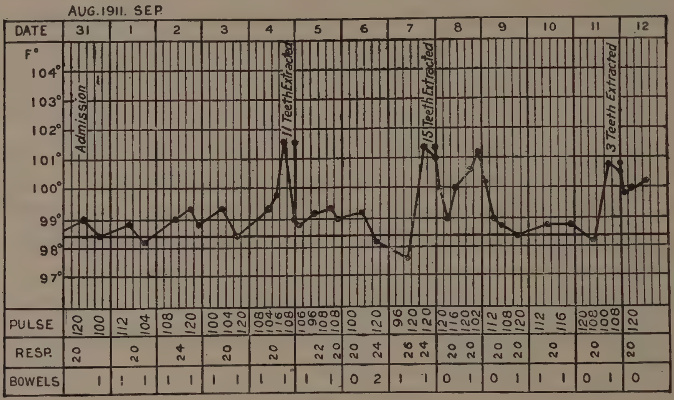

FIG. 678.

One is well aware that treatment by free extraction of all teeth which are the source of sepsis, as suggested above, is opposed to the teachings of many practitioners. With those who hold that local treatment will often result in a removal, or at least an improvement in the general condition, one fully agrees, but—and this is the important fact—the source of sepsis has not been eradicated. The sepsis may possibly be lessened in degree, but nevertheless it remains and must continue to damage the tissues slowly but surely. The damage to the tissues is progressive, and when, through the loss of the teeth, a natural cure is effected, the damage of the tissues may be irreparable. Dental practitioners cannot be justified in allowing such a condition to be brought about, and it is our duty to place clearly before the patients the risks they run.

(I) SEQUELÆ.

The various local and general sequelæ that may arise from the presence of infection in the mouth are discussed in Chapter XXV. But there is one point upon which particular stress must be laid here, and that is that the *absence of objective symptoms is no proof that damage—and possibly irreparable damage—is not in progress.* Patients will harbour infection in their mouths for years without apparent harm, but eventually symptoms are almost certain to appear. The following case is typical :—

A man, aged about thirty-six, at the beginning of 1909 developed a swelling in the left maxilla, and examination of the mouth showed slight necrosis in this region with parodontal disease accompanied by much sepsis, necessitating the removal of all the teeth. His previous history was as follows : His normal weight was about thirteen stone, he was an exceptionally fine type of man and had succeeded in carrying off many prizes in important athletic contests.

About the beginning of 1907 he discovered that he was losing weight, and in February 1909 he weighed only about nine stone. During this period his mouth had, he said, caused him very little trouble, except that he had neuralgia occasionally, and his teeth had from time to time become loose. All his teeth were eventually removed, and a rapid improvement in his general condition followed. On questioning this patient a very definite history of parodontal disease spreading over a dozen years was disclosed.

This patient had, unknown to himself, been the subject of parodontal disease for years, and for a long period he had been able to combat the absorption of the toxic matter, probably by the production of antibodies, and so remain immune. After a time this immunity had broken down and the toxic material became rapidly destructive to the tissues.

CHAPTER XIX

Saliva.[1]

Physical and Chemical Characters of Mixed Saliva—The Individual Secretions—Factors affecting the Nature of the Saliva—The Functions of Saliva—The Nervous Control of the Secretion—Drugs affecting the Flow of Saliva—Therapeutical Applications and Modes of Administration—Bactericidal Properties—Relation of the Saliva to Caries of the Teeth—Salivary Calculi—Degeneration of the Salivary Glands—Infection of Salivary Glands.

(A) PHYSICAL AND CHEMICAL CHARACTERS OF MIXED SALIVA.

Saliva is the mixed secretions from the three pairs of salivary glands (parotid, submaxillary, and sublingual) and the numerous small buccal mucous glands scattered over the whole of the oral mucous membrane, and particularly over the posterior aspect of the hard palate, where their massing is sometimes described as the palatal salivary gland. As will be described later on, it is possible to collect separately the secretion of each gland and examine them as such, but it is obviously best to consider the mixed secretions, since it is these with which we have to deal in relation to speech, food, mastication, deglutition and digestion.

The Physical Characters of Mixed Saliva are :—

It is a cloudy, colourless, slimy fluid, of specific gravity 1002–1008. The cloudiness is partly due to the presence of desquamated epithelial cells, clumps of colloidal mucin, leucocytes, and other debris.

In reaction saliva is just faintly acid, with a pH of from about

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[1] For the revision of Sections A to G of this chapter the authors are indebted to W. A. M. Smart.

6·4 to 7·0. About 0·6 per cent. of its weight consists of solids. In quantity it is estimated that at least one litre is produced per twenty-four hours. In the herbivora the quantity amounts to many litres.

THE CHEMICAL COMPOSITION OF SALIVA is as follows :—

(1) Inorganic Salts.—Sodium chloride, with calcium and magnesium carbonates and phosphates. The total amount of these salts is about 0·2 per cent. There is also present a trace of potassium thiocyanate (sulphocyanide) though its significance is not understood. These salts are present in ionised form.

On standing or heating saliva loses carbon dioxide, which causes the deposition of a cloudy precipitate of calcium carbonate, which helps to form the incrustations of tartar on the teeth.

$$Ca(HCO_3)_2 \rightleftarrows CaCO_3 + CO_2 + H_2O.$$

The same equation also holds for the 'furring' of kettles and boilers by hard water. Calcium biphosphate $Ca(H_2PO_4)_2$ is also held in solution so long as it is acid. When the saliva becomes alkaline the normal calcium phosphate is precipitated.

$$3Ca(H_2PO_4)_2 \rightleftarrows Ca_3(PO_4)_2 + 4H_3PO_4.$$

(2) Organic substances to the extent of 0·4 per cent. consisting mostly of coagulable protein, mucin, urea, uric acid, and a diastatic enzyme, ptyalin.

(B) THE INDIVIDUAL SECRETIONS.

THE INDIVIDUAL SECRETIONS OF EACH GLAND may be briefly summarised as :—

(*a*) *The Parotid glands* give a clear watery serous secretion, the poorest in solids, and containing no mucin. This secretion is estimated to contain four times the concentration of ptyalin (amylase) found in the mucous salivary glands.

(*b*) *The Submaxillary* (*submandibular*) *glands* are mixed serous and mucous, the former predominating. Their secretion is the richest in solids.

(*c*) *The Sublingual glands* also give a mixed mucous and serous secretion. In this case the mucous secretion predominates; indeed, it is this mucin which makes the saliva so viscid.

(C) FACTORS AFFECTING THE NATURE OF THE SALIVA.

Factors affecting the Nature of the Saliva secreted.

As might have been anticipated, diet has a profound effect on the physical and chemical qualities of the saliva. Other factors also have an influence, though much less in importance.

In the past investigations have been mainly carried out on mammals—usually dogs with artificial salivary fistulæ. The surgical technique is simple. Stenson's duct is brought to the exterior of the cheek and sewn into the incision. There is no inconvenience to the dog, and samples of saliva may be collected at any time. This method was first used by de Graaf and improved upon by Pavlov.

It was soon found that secretion is not continuous, and is dependent on the nature of the food. With meat there is but a scanty secretion, but with dry food the flow at once becomes more dilute and greater in volume. Experiments on man did not come along until comparatively recently.

(1) Diet and its effect on human salivary secretion was specially investigated by Pickerill.[1] He devised a special technique for the collection of saliva from Stenson's duct during the mastication of various kinds of food. Every effort was made to carry out the experiments under identical conditions. Controls were carried out with resting saliva ; that is, saliva present in the mouth some hours after eating. He found that with a fruit stimulus the total alkalinity is greater than with a carbohydrate stimulus. Variations in the total output of saliva were similar to the variations in total alkalinity. Acid substances evoked the greatest amount of ptyalin per minute, while alkaline dentifrices had a marked depressing effect both on the amount of secretion and the degree of alkalinity.

Neilson and Terry[2] showed that in dogs the amylolytic power of the saliva of bread-fed dogs was greater than that of meat-fed dogs.

Champion in 1932[3] showed that the reaction of human saliva varied directly with the hydrogen ion concentration. He found that the *p*H is not affected by substances in the mouth

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[1] 'Dental Caries.' Baillière, Tindall and Cox.

[2] *Amer. Journ. of Physiol.*, xv, 406.

[3] *Brit. Dent. Journ.*, 1932, liii, 209.

(acid or alkali), but masticatory movements cause a rise in $p\mathrm{H}$; and that it is not influenced by the ratio of carbonic acid to bicarbonate in the blood even when acid or alkali salts are ingested.

(2) Drugs, as will be seen in the next section, can greatly affect the rate of salivary secretion. The qualitative effects have not been fully investigated.

(3) Age.—Ptyalin is present in premature infants, gradually increasing up to six months after birth.

(4) Psychic factors are well known to affect the nature and amount of the saliva. In fear (adrenaline stimulation) the saliva becomes very viscid and scanty—'my tongue cleaveth to my jaws.'[1] On the other hand, the smell and sight of food, talking about it, or hearing the preparations for a meal (the rattling of plates) will readily induce a secretion, as we know from observations not only on animals but also on ourselves.

(D) THE FUNCTIONS OF SALIVA.

The Functions of Saliva depend on its physical and chemical properties.

(*a*) **Its physical character,** especially its slimy mucous nature, enables the oral cavity to remain moist in spite of constant evaporation, and also facilitates speech, mastication, and the lubrication of a bolus of food, ready to aid the œsophageal muscles in the readier passage of food to the stomach. The watery part of the saliva dissolves the soluble portion of the food, provided it stays long enough in the mouth (for some people 'bolt' their food). Moreover, it dilutes irritant and hot foods.

(*b*) **Its chemical character** is due to the presence of an enzyme known as *ptyalin*, whose particular or specific function is to hydrolyse the large starch molecules, which constitute so large a part of carbohydrate food, into maltose, a disaccharide. This reduction is carried out in stages, first to soluble starch, erythrodextrin, achroo-dextrin, and lastly to maltose. The ptyalin therefore converts the insoluble and unabsorbable starch molecules into maltose, a soluble and smaller molecule but still unabsorbable. For this reaction the starch granules have to be ruptured as they are in cooking, otherwise they are protected from action by their resistant cellulose cell wall.

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[1] Psalm xxii, 15.

Oral and Gastric Digestion of Carbohydrates.—It must not be supposed that salivary digestion goes on to any extent in the mouth, for its stay there is not long enough. In the stomach the gastric juice only slowly penetrates the mass of food lying there. During this time, for say roughly half an hour, salivary digestion still proceeds, but only down to maltose stage. There is an enzyme present in pancreatic juice, called amylase, which has the same end result as ptyalin, in that all starchy food is reduced to the stage of maltose. Further reduction to the monosaccharide condition, glucose, is carried out by the intestinal juice.

(E) THE NERVOUS CONTROL OF SALIVARY SECRETION.

The Nervous Control of Salivary Secretion is of a reflex nature. We have therefore to consider the component parts of this reflex, that is, the afferent nerves, the salivary centres, and the efferent nerves.

(1) The Afferent nerves consist of the following sets :—

(*a*) *Sensory nerves* in the oral mucous membrane conveying ordinary sensation (particularly heat and cold), but more especially those of taste, proceeding from the taste buds in the circumvallate papillæ. These afferent origins are stimulated by the presence of dissolved food or drugs in the mouth. The fibres belong to the fifth and the ninth cranial nerves.

(*b*) *The Vagal nerves* in the gastric mucous membrane may be irritated by the presence of unsuitable food or drugs in the stomach leading to nausea and even vomiting—reflexes associated, among other things, with increased salivation.

(*c*) *Psychic stimuli* are nearly always involved—the mere anticipation of food. Thus the sight of a dainty meal, the smell of hot coffee or soup, and even the clink of glasses may lead to an outflow of saliva. Hence the first, second, and eighth cranial nerves must therefore be added to the list of afferent fibres.

(2) The Salivary Centre consists of a group of cells situated in the formatio reticularis of the pons between the nucleus of Deiters and that of the seventh cranial nerve.

There are also peripheral ganglia which act as subcentres—the otic ganglion for the parotid, and the submaxillary for the other two glands.

(3) The Efferent nerves consist of fibres from the Autonomic system :—

(*a*) *The Parasympathetic supply.*—That for the submaxillary and sublingual glands is composed of fibres which run from the centre via the chorda tympani branch of the seventh nerve, and then join the lingual branch of the fifth nerve.

Those for the parotid gland consist of fibres running in the auriculotemporal nerve, and probably derived from the ninth nerve via the otic ganglion.

Stimulation of these nerves leads to a local vasodilatation and increased salivary secretion.

(*b*) *The Sympathetic supply* comes from the plexus on the external carotid artery. On stimulation there is a scanty secretion of viscid saliva, accompanied by local vasoconstriction.

It is not definitely known whether each alveolus has a double nerve supply, or whether parasympathetic and sympathetic fibres proceed each to different alveoli, but opinion inclines to the latter possibility. Our knowledge of the precise part played by the sympathetic system on the salivary secretions is not yet fully determined. In the case of the parasympathetic supply it is considered that the effects of its stimulation lead to a freeing of a substance, probably acetylcholine, whose purpose is to set in motion the receptor neuroacinar mechanism controlling salivary secretion.

(F) DRUGS AFFECTING THE FLOW OF SALIVA.

Drugs which act upon the Salivary Glands.—It has just been explained that salivary secretion is a reflex act. So far as drugs are concerned it is therefore possible for them to act upon any or all of the component parts of the reflex arc—the afferent sensory origins, the centre (or centres), and the efferent nerve ends. The effects of such action may be an increase or decrease in the amount and possibly the character of the saliva secreted, dependent of course on the particular drug employed. Those which increase the rate of salivary flow are known as *sialogogues*, and those which diminish the rate are *antisialogogues*. It is also possible for a drug to act directly upon the alveolar cells themselves. Hence we have the following scheme :—

(1) **Drugs affecting the afferent nerve origins** in the mouth and stomach. Bitter, aromatic, pungent, and acid substances produce an increase in the rate of secretion. On the other hand, demulcents such as gum acacia, tragacanth, and glycerine, and astringents like alum, zinc sulphate, and tannic acid, lead to a diminished secretion.

(2) **Drugs which act on the salivary centre and ganglia** are not used in dental practice. The nicotine group (nicotine, lobeline, and coniine) first stimulates secretion, when used in small doses, and paralyses it when in excess. Cigarette and pipe smoking cause a free flow of saliva in some people. Morphine or opium decreases the flow, probably by some central action. The dry mouth after a morphine sleep is well known.

(3) **Drugs affecting the efferent nerve ends** are of three types :—

- (*a*) Those which irritate the parasympathetic supply, the acetylcholine group being the best example. This includes physostigmine (eserine), pilocarpine, acetylcholine, and muscarine. Of these only the first two are in general use.
- (*b*) Those which impose a block on parasympathetic action, such as the various members of the atropine group—atropine, hyoscyamine, hyoscine, and stramonium. Of these only atropine is used—in cases of marked salivation.
- (*c*) Those which irritate the sympathetic supply. This includes the adrenaline group—adrenaline, ephedrine, and several others. They are of no clinical value.

(4) **Drugs affecting directly the alveolar cells.**—It has been found that certain drugs after absorption into the blood are excreted in part by the salivary glands, and in so doing stimulate them to an increased rate of secretion. Examples are the iodides, the salicylates, and mercury salts, more particularly the latter. Histamine is a good siálogogue, perhaps by direct action on the cells.

Summing up the above in a more practical manner, we have :—

Sialogogues—organic acids (citric and tartaric), pilocarpine, aromatics, alcohol, ether, and emetics (in subemetic doses).

Antisialogogues—atropine, tannin, alkalies, and morphine.

(G) THERAPEUTICAL APPLICATIONS AND MODES OF ADMINISTRATION.

The Therapeutical Applications of the above-mentioned drugs are made in the case of excess or scanty secretion of saliva.

(1) Hyposecretion or Xerostomia (*i.e.* dry mouth) may occur in certain cases, especially after the administration of morphine (opium) or any member of the atropine group. It may sometimes be seen in senility, and is often an accompaniment of feverish conditions, and occurs in a particularly distressing form after exposure of the salivary glands to the action of radium emanations. For the relief of this condition one should prescribe sialogogue collutoria or dentifrices.

(2) Hypersecretion amounting to constant dribbling, and highly disconcerting to the patient, may occur in early pregnancy, in certain types of insanity, and in mercurial poisoning. In these circumstances the use of an astringent such as krameria, tannic acid, or a drug like atropine (in $\frac{1}{100}$ grain doses) to semi-paralyse the secretion, is indicated.

The Modes of Administration may be either by the use of collutoria (mouth washes) or dentifrices (tooth powders and tooth pastes). These preparations are of course varied for many different purposes—deodorant, antiseptic, anodyne, sialogogue or antisialogogue, as the case may be.

Collutorium Astringens.

Tinct. of krameria	30♏
Liquefied phenol	12♏
Oil of peppermint	3♏
Alcohol (90%) to	1℥

A teaspoonful to be added to a tumblerful of water for use.

Collutorium Stimulans.

Sodium citrate	1℥
Sodium bicarbonate	1℥
Sodium chloride	½℥
Oil of cassia	5♏
Oil of peppermint	10♏

Half a teaspoonful to be dissolved in a small tumbler of warm water for use.

Thus we increase salivation by the use of organic acids, like citric or tartaric. Lemonade does very well, especially if it contains carbonic acid. Some prefer pyrethrum or saccharine. If we want to diminish the flow, then an astringent, usually some member of the tannic acid series, is indicated.

(H) BACTERICIDAL PROPERTIES.

Sanarelli[1] states that human saliva possesses the power of destroying micro-organisms when their number is not considerable, and even when it fails to destroy them, as in the case of the bacillus of pneumonia, it seems capable of modifying their normal characteristics by weakening them, and then rendering them completely inactive. Sanarelli experimented chiefly with the more common of the micro-organisms which are found in the mouths both of healthy and unhealthy individuals, namely, the *Staphylococcus pyogenes aureus*, *Streptococcus pyogenes*, the bacillus of diphtheria, the *Micrococcus tetragenus*, the diplococcus of pneumonia, the typhoid bacillus, and the cholera spirillum. The saliva was filtered with a Chamberland filter and experiments were done by the plating method.

Hugenschmidt[2] records numerous experiments with filtered saliva, and he has come to the conclusion that 'the bactericidal action of the saliva appears to be very problematical.' He was only able to detect it on the torula and *Staphylococcus aureus*. The saliva, he considers, has an important mechanical action, as it dilutes the bacteria, glueing them together so that they are swallowed and destroyed in the stomach by the action of the gastric juices. In connection with these experiments it has been pointed out by Miller that the effect of filtering the saliva is to deprive it of the greater part of its nutrient matter, so that it contains but a small fraction of the organic matter usually present in culture media. The refusal of bacteria to grow in such a fluid is possibly due to inanition and cannot therefore be regarded as a proof of antiseptic action.

In a series of experiments carried out by Miller[3] this source of error was excluded. Bouillon of double strength, made from beef extract, peptone, and sugar, was prepared, and 5 c.c. placed in three tubes, A, B, and C. To tube A, 5 c.c. of the saliva of a person immune to caries were added; to tube B, 5 c.c. of the saliva of a person not immune; and to tube C, the control, 5 c.c. of water. By this means the strength of the bouillon was reduced to the normal. The saliva was filtered through a Chamberland filter. The three tubes were inoculated with a bacterium obtained from carious dentine and incubated. As a result of this experiment Miller found that the saliva exerted no retarding influence on the growth of the organism.

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[1] *Centralblatt für Bakteriologie*, x, 817.

[2] *Dental Cosmos*, October 1896.

[3] *Dental Cosmos*, 1903, xlv, 1.

Experiments were then undertaken to test the rate of acid production. Bouillon, made of beef extract, peptone, and sugar, of five-fold strength, was taken, and to one part of bouillon four parts of each saliva were added. The tubes were then inoculated and the rate of acid production determined by the amount of a 5 per cent. solution of bicarbonate of soda required to neutralise the amount of acid formed. One experiment gave the following results :—

Relative Amount of Acid Formed.

	In 42 hours.	In 62 hours.	Total.
Saliva of A	9·5	5·5	15·0
,, B	9·0	7·0	16·0
,, C	10·0	7·5	17·5
Control water ...	9·0	7·0	16·0

The amount of acid produced in the control tube containing water and the three tubes containing saliva showed no appreciable variation. Further experiments were undertaken on the undiluted saliva, non-altered saliva, etc., with similar results, and the conclusion Miller came to was 'That mixed human saliva—whether filtered or unfiltered, in its normal state or condensed by evaporation over the water-bath, or at the temperature of the human body—does not possess the power to prevent or retard processes of fermentation or putrefaction.'

Protective properties have been accredited to the buccal mucus. Miller, however, came to the conclusion that 'growths of bacteria and fermentation and putrefaction processes take place in oral mucus quite as readily, if not more so, than in the mixed saliva of the same person.' He says that 'it seems hardly probable that the buccal mucus can contain any bactericidal property in view of the fact that the mucous membrane of the mouth is invariably found to be covered with large masses of bacteria.' On the other hand, the action of mucus in other cavities of the body must not be lost sight of. Wurtz and Lermoyez have shown that 'after three hours' contact with the nasal mucus at 38° C. the spores of the *Bacillus anthracis* are killed, while the *Staphylococcus aureus*, the streptococcus and the *B. coli* are greatly attenuated.' Thomson and Hewlett[1] also found the nasal mucus possessed a retarding influence, but no bactericidal property.

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[1] *Lancet*, 1897, 860.

The mucus secreted normally by the cervix uteri destroys and attenuates the germs of the vagina and prevents them from penetrating into the uterine os ; still further, in connection with the bronchi, Widal considers that ‘ the innumerable glands which exist on the surface of the respiratory mucous membrane secrete constantly a mucus which has a bactericidal action.’ Arguing by analogy, it would certainly seem probable that the mucus in the mouth when healthy does exert a protective influence against infection.

It is probable that *phagocytosis* plays an important rôle in protecting the oral cavity against pathogenic organisms. Hugenschmidt refers to the chemiotactile properties of mixed saliva, and shows that non-filtered human saliva possesses positive chemiotactile properties. This he demonstrates by the following experiments : Human saliva ejected in the morning was left for some time in an experiment glass ; the upper part of the liquid, which had become clear, was then introduced in small quantities into capillary tubes which were each closed at one extremity ; the tubes were then inserted into the peritoneal cavity of a guinea-pig and left for eight hours, after which they were removed, and, on being examined, leucocytes were found to have formed a dense plug 2 mm. long. In another experiment, in which the saliva was kept for twenty-four hours in an oven at 37° C., and in which the number of organisms had greatly increased, the plug formed by the leucocytes in the capillary tube was visibly larger, thus showing that the attraction exerted by the toxins is in relation to the intensity of the culture, and consequently with the quantity of the bacterial products present in the liquid. From these experiments it may be inferred that when there exists in the interior of the alveolar process after an extraction, or in any other part of the buccal parieties, a cavity where the saliva can remain and become the medium of an abundant culture, the saliva presents chemiotactile properties in relation to the quantity of organisms which have developed in it. The leucocytes of the neighbourhood will be energetically attracted towards the part, and will, in a very marked way, accomplish their protective action.

Experiments in which the saliva of a guinea-pig was introduced into the same animal’s peritoneal cavity produced the same results as human saliva, thus demonstrating the fact that the saliva of one animal attracts the leucocytes of the same animal. After it had been proved that saliva possessed positive chemiotactile properties, experiments were made with a view of ascertaining whether the phagocytes of an animal are capable of ingesting and digesting the organisms which occur in the salivary secretions. That the phagocytes of an animal are capable of doing this is shown by the following experiments :—

Human saliva which had been allowed to stand and clarify was mixed with leucocytes from guinea-pigs. The mixture was placed in an oven at 370° C. for an hour, after which it was placed on slides, fixed, and coloured by Ehrlich's process. Microscopical examination of the slides showed that the leucocytes had taken up the organisms with great avidity. Similar experiments with the same animal gave similar results. In another experiment recorded, the activity of the destruction of the organisms by the phagocytes is clearly demonstrated. 'A trace of saliva was taken from a guinea-pig and from this preparations were made and stained, some with methyl blue and others by the Gram method. This done, a wound was made in the gum and in the median line of the mandible, the mucous membrane being removed and the bone scraped. Twenty-four hours later the wounds presented a white coating consisting of leucocytes, almost all polynuclear, some mononuclear. These leucocytes, by proper stainings, were found to contain an abundance of microbes similar in form and character of stain to those seen in the preparation of the saliva. A considerable number of the englobed microbes were less energetically stained than those from the surrounding liquid, thus demonstrating their degeneration.'

In the experiments above recorded the saliva was used un filtered and therefore mixed with organisms. In order to test whether filtered saliva possessed these chemiotactile properties, the following experiment was carried out : Saliva filtered through a Chamberland filter was introduced into six capillary tubes each of which had one end closed. In six other tubes cholera culture was introduced, and into another six physiological serum. These three bunches, separately attached, were placed in the peritoneal cavity of a guinea-pig and left for ten hours. Examination at the end of that period showed dense plugs of leucocytes in the tubes containing cholera culture, but few leucocytes in the tubes containing filtered saliva, and no trace of any in the tubes containing physiological serum. 'It would thus seem that the positive chemiotactile properties of mixed saliva depend upon the presence of micro-organisms.'

But phagocytosis is probably not the only means by which the oral cavity is rendered immune to pathogenic organisms, for it is probable that the epithelial cells and also the vital antagonism of the microbes lend their aid. The epithelial cells of the oral cavity are constantly being replaced, this desquamation being especially active during the act of mastication. Although the epithelial cells do not possess phagocytic properties, nevertheless they have attached to their surface, and are also to a certain extent penetrated by, scattered bacteria. These cells on being dislodged are carried away with the organisms to the stomach, where the latter are destroyed.

Another factor to be considered in connection with the immunity of the oral cavity to infection may be the antagonism of organisms to one another, the organism not common to the mouth succumbing to those normally present. The following experiment, carried out by Miller on himself, throws light on this question. He thoroughly rinsed his mouth with a bouillon culture of *Bacillus prodigiosus*[1] containing over 2,000,000,000 bacilli. The number of bacilli in a loop of his saliva was ascertained at the beginning of the experiment, and at the end of one, two, three, and six hours, with these results :—

Bacteria at beginning	97,600
,, after 1 hour	1,220
,, ,, 2 hours	127
,, ,, 3 ,, ...	17
,, ,, 6 ,, ...	0

His mouth at the end of three hours was practically free from these organisms. The elimination of pathogenic organisms such as those of tubercle, typhus, etc., which are constantly finding an entrance to the mouth, may be brought about by a struggle with the normal flora of the cavity.

To sum up, the facts quoted above would seem to indicate that the soft tissues of the mouth are rendered immune :—

(1) By the process of phagocytosis.

(2) By the normal functional activity of the mouth, causing desquamation of the epithelial cells with the adherent bacteria, the latter being destroyed by the gastric juice.

(3) By the mutual antagonism of the micro-organisms.

(4) By the hypertonicity of saliva which in itself attracts leucocytes when it comes into contact with a raw surface, just

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[1] The bacillus of 'bleeding bread.'

as slightly hypertonic sodium chloride solution does (*cf.* the licking of wounds by animals).

(5) The blood supply.

(I) THE RELATION OF THE SALIVA TO CARIES OF THE TEETH.

A free flow of saliva by cleansing the teeth of food debris lessens their liability to caries. The phosphates probably have a protective action upon the teeth. Lactic acid 'readily combines with phosphates to form harmless lactates, and the lactic acid will be more readily and rapidly satisfied if the phosphates are in solution than if they are in a solid and extremely hard mass like enamel.' 'Acid phosphates may be formed, but there is no evidence to show that these, under such circumstances, can exert any effect upon the enamel surface.' [1]

The recent (1930) researches of J. R. Marrack showed that the saliva of children in whose mouths dental caries is prevalent contains a smaller percentage of calcium than that of the same children several weeks after the carious teeth had been treated. No explanation of this phenomenon is given, but it seems probable that the irritation of carious teeth may stimulate an increased flow of saliva which is therefore poorer in its solid—and hence its calcium—content. The truth of this might easily be tested by comparing the calcium content of the saliva of caries-free individuals under normal circumstances with that from the same individuals after it has received prolonged stimulus, such, for instance, as that obtained by chewing gum, or sucking acid drops ; or by comparing the saliva of edentulous persons before the insertion of dentures with that during the two or three days subsequent to their insertion.

Ptyalin, in Pickerill's opinion, is an important protective agent for the teeth. It converts the insoluble and adhesive substances, such as starch, into a soluble and easily removable substance (maltose), and, provided that there is a free flow of saliva, stagnation is obviated and circulation promoted. He considers that a small amount of ptyalin is harmful. His argument is as follows :—

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[1] The method adopted to estimate the phosphate was 'that of titration with N/5 uranium nitrate. The saliva was diluted, and there was added a small quantity of sodium acetate acidified with acetic acid. The indicator used was potassium ferro-cyanide' (Pickerill).

(i) In the conversion of starch into maltose by the action of ptyalin, erythro-dextrin and achroo-dextrin are formed as intermediate products.

(ii) The rapidity of action depends upon the amount of the ptyalin and its concentration.

(iii) If the ptyalin is present in considerable amount the conversion into maltose is rapid, and it is quickly removed by the flow of saliva.

(iv) If the ptyalin is small in amount, the dextrin formed will remain longer and with the maltose cling to the teeth. The mouth organisms will convert the maltose into lactic acid, and this in its turn will destroy the ptyalin, with the result that the 'starch is literally gummed to the teeth, to be slowly but surely transformed into lactic and other acids.'

The effect of mucin may be harmful or beneficial. When partly dissolved it is very viscous, and in this condition is harmful, as it tends to bind food debris to the teeth and so promotes stagnation. Acid substances precipitate mucin, and alkaline salts are taken down in the precipitate, and the teeth are coated with a thin organic alkaline film. The mucin is acted on by bacteria, and alkaline products are formed which tend to neutralise any acid formed by fermentation.

The relation of potassium sulphocyanide to caries has been freely discussed. It is maintained by certain authors that the presence of sulphocyanides diminishes the liability to caries. The amount of potassium sulphocyanide in the saliva is very small, varying from 0·0075 per cent. to 0·03 per cent. Miller[1] has shown that potassium sulphocyanide in the strength met with in saliva has no retarding influence.

(J) SALIVARY CALCULI.

A calculus occasionally forms in the ducts of the salivary glands. It may form in the substance of the gland, but this is rare, as a deposit formed in this situation is usually washed into the duct. If the deposit is situated in the substance of the gland, it may give rise to serious inflammatory trouble ; the flow of saliva from the duct of the gland is obstructed, and the trouble may lead to abscess or even destruction of the gland as ascending infection of the duct takes place.

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[1] *Dental Cosmos*, January 1, 1903.

A calculus is more frequently met with in the submaxillary ducts than in the parotid duct, and this is possibly due to the mucoid character of the submaxillary secretion, the fact that it contains a higher percentage of solids, and to the position of the duct rendering it more liable to injury.

Symptoms.—When a salivary calculus is deposited in the duct of a gland, the obstruction, which may be either as small as a millet seed or larger than a filbert, will give rise to a swelling of the duct and gland. Salivary calculi situated in the floor of the mouth may simulate ranula, from which they should be diagnosed by noting that in the case of salivary calculus the flow of saliva is stopped, and that a small probe cannot be passed along the duct, while careful digital examination will reveal a hard concretion in the duct.

At times the presence of a salivary calculus in Wharton's duct will give rise to appearances and symptoms suggestive of malignant disease. In one case seen, the floor of the mouth was much swollen, the tongue pushed upwards, and there was a fungating excrescence surrounded by tissue in an indurated condition. The tissues covering the mandible and the neck were much swollen, and the lymphatic gland lying in the submaxillary region enlarged. The patient presented a cachectic appearance. The whole condition seemed typical of malignant disease.

In a case recorded by T. E. Constant, a calculus in the parotid duct caused symptoms simulating alveolar abscess.

Origin.—The cause of the formation of salivary calculi is not clear. It has been suggested that they arise in a manner similar to other calculi, by precipitation and secretion of the salts around a foreign body. This view is supported by the occasional presence of a nucleus of foreign matter within a salivary calculus. A minute foreign body can easily enter the main salivary ducts and form a nucleus.

Bland-Sutton [1] considered that the principal conditions determining the formation of gall-stones are catarrh of the epithelial lining of the gall-bladder plus the presence of micro-organisms, and it is possible that salivary calculi may arise in a similar way, *i.e.* from an ascending infection.

Size.—Salivary calculi vary considerably in size. They may weigh as much as 25 gr., and the fusiform-shaped calculi reach 1 in. in length.

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[1] 'Gall-stones and Diseases of the Bile-ducts.' Nisbet, 1907.

Treatment.—Treatment consists in making an incision over the hard swelling and removing the calculus. It is best to secure the concretion in a fixed position before making the incision. Cases remaining untreated may give rise to suppuration, either in the region of the duct or in the gland itself. The abscess at times opens externally and thus gives rise to a salivary fistula. Salivary calculus as a deposit on teeth will be considered in the following chapter.

A case in which six calculi were found in the parotid duct in a child aged thirteen is recorded.[1]

Differential Diagnosis.

With regard to the differential diagnosis, it is convenient here to consider the condition known as ranula.

Ranula (diminutive of rana = a frog). This is a cystic swelling containing a glairy mucoid fluid occurring in the floor of the mouth, usually unilateral and sometimes originating on the under surface of the tongue. There are two varieties. *The simpler form* is a myxomatous degeneration of a mucous gland, either one of those situated in the floor of the mouth or the gland of Blandin and Nuhn ; it shows as a transparent cystic swelling having small vessels traversing its surface, and Wharton's duct may also cross it. *A deep form* probably originates from an unobliterated cervical sinus and may therefore be regarded as a distension and displacement of a lateral cervical dermoid. Either may become the size of a pigeon's egg and is painless.

Treatment consists of complete extirpation by dissection ; this, however, is not always possible in the deep forms, when as much as possible should be removed by dissection and the wound packed and allowed to heal by granulation.

(K) DEGENERATION OF THE SALIVARY GLANDS.

Lymphoid degeneration of the salivary glands has been described by W. S. Handley.[2] The change consists in the total destruction of the epithelial elements of the gland, accompanied by hypertrophy of the lymphoid elements. The microscopical structure of the gland becomes practically indistinguishable from that of the tonsil or of a lymphatic gland, and the saliva-secreting function of the gland is destroyed.

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[1] *Amer. Med. Surg. Bull.*, July 25, 1897.

[2] *Trans. Odonto. Soc.*, xxxix, 186.

The clinical features of the condition are pain, tenderness, and swelling of the gland, accompanied by a mucoid discharge. A small calculus is sometimes present in the duct. In the cases recorded, the primary change would appear to be chronic catarrh of the salivary duct due to bacterial invasion from the mouth. The most satisfactory treatment is excision of the gland.

A case of purulent discharge from both parotid glands has been placed on record by C. S. Tomes.[1] The trouble dated from an attack of typhus thirty years previously. The discharge was indistinguishable from pus, but its power of affecting starch had not been destroyed.

(L) INFECTION OF THE SALIVARY GLANDS.

Infection around the orifices of the ducts of the salivary glands may spread through the ducts to the substance of the glands. The condition starts by a thickening of the papilla guarding the orifice of the duct; the duct becomes a thickened and painful cord, and dilates during secretion. If the gland substance becomes involved there is considerable swelling and tenderness, which is increased during salivary secretion. A calculus may form in the duct; the calculus if small has a tendency to alter its position, but if large it is stationary. The calculus is always the seat of great tenderness. The condition is usually asymmetrical; when symmetrical it may simulate mumps.

Septic conditions of the mouth are always present. Removal of the sepsis is usually followed by a relief of the symptoms, but in cases of long standing it may be necessary to slit up the duct or even remove the gland.[2]

Acute infection notably of the parotid gland may occur in the course of acute illnesses or after severe operations. It is a severe disease and frequently has a fatal termination; it is not very often seen now, as dental treatment before operations is far more universally practised than heretofore. (See p. 991.)

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[1] *Trans. Odonto. Soc.*, 1891, 32.

[2] Spencer, W. G. *Brit. Med. Journ.*, January 28, 1922.

CHAPTER XX

Deposits on and in Teeth.

Salivary Calculus—Serumal Calculus—Stains.

(A) SALIVARY CALCULUS.

Calculus derived from the saliva may be deposited on the teeth or artificial dentures. This form of calculus is familiarly known as 'tartar.'

(1) Situations.—The lingual surfaces of the mandibular incisors and canines and the buccal surfaces of the maxillary molars are the most common situations of these deposits; it will be noted that these surfaces are immediately opposite the orifices of Wharton's and Stenson's ducts respectively.

(2) Varieties.—Clinically there are two varieties, 'soft' and 'hard.' The distinction is purely arbitrary. The difference depends upon the rate of deposition. *Rapidly deposited tartar* is soft in character, buff-coloured, and occurs in large quantities. *Slowly deposited tartar* is dark and hard in character, and is exceedingly tenacious. The colour of the tartar is affected by conditions which produce staining, such as tobacco smoking, the drinking of strong tea, etc.

(3) Conditions Favouring its Deposit.—The deposition of salivary calculus is assisted by: (*a*) Rough surfaces, as in 'hypoplasic' teeth; (*b*) the presence of foreign bodies, such as dentures which are not kept clean; (*c*) lack of function, which does away with the self-cleansing of the act of mastication. Any condition which interferes with the proper functioning of the teeth favours the accumulation of salivary calculus. Such conditions may consist of the presence of some pathological process or its result and is usually one of a sufficiently painful or uncomfortable nature to throw certain teeth, usually those of one side only, out of action, and so deprive them of their normal friction with the coarser particles of food during the act of mastication. Such process may consist of an inflamed or exposed dental pulp, local periodontitis, either acute or chronic, a 'polypus' of the pulp or gum, gingivitis, the loss of antagonising teeth on that side, or malocclusion on one side only.

Apart from the above, some people acquire the habit of using one side only in masticating; obviously also the use of soft foods and lack of the use of the brush will favour its deposition and, in addition, there are not infrequently cases of certain individuals who, apart from any of the above causes, are particularly prone to the deposition of salivary salts on their teeth; in such cases it is probable that the saliva itself is abnormal, possibly with regard to its solid content, but further investigation of these cases is needed. F. W. Broderick considers that a condition of alkalosis very much favours the deposition of salivary calculus on the teeth.

(4) Mode of Deposit.—The deposit, which may attain considerable proportions, starts at the cervical margin and, as it increases, tends to assume a wedge shape, the base being towards the gum. The friction of mastication removes the calculus and the disuse of the teeth favours its deposition.

The calculus is formed by precipitation of the salts from the saliva. Calcium carbonate and calcium phosphate are insoluble in pure water, but soluble in water containing carbonic acid gas. When the saliva reaches the oral cavity, the carbonic acid gas passes out of solution and the lime salts are precipitated.

The researches of A. Bulleid show that deposition of salivary calculus is intimately associated with the presence of *Leptothrix buccalis*, and that it does not occur in its absence; indeed, salivary calculus has been defined as 'a petrified mass of Leptothrix.'

In the opinion of H. Prinz [1] the formation of salivary calculus is of the nature of a physico-chemical process and depends upon the co-ordination of three basic factors: (*a*) The presence of a super-saturated solution of calcium salts in a colloidal medium, *i.e.* the saliva; (*b*) its stagnation in a sheltered place; and (*c*) a parental or foreign inanimate substance having a hard, rough surface which serves as a nucleus for its attachments. 'The stagnated, mixed saliva, in conformity with Gibb's law of surface tension, concentrates its colloids upon its own surface. At first a faint nubecula appears which by its own concentration slowly forms a dull lustrous pellicle. Simultaneously the solvent power of the saliva is thereby physically diminished, thus liberating the crystalline calcium salts, which are at once caught by the freshly formed colloidal stroma. The pellicle gradually becomes specifically heavier than the surrounding fluid and slowly sinks or is forced along the line of least resistance by the movements of the cheeks, lips, and tongue until it meets with some obstruction. Any sheltered surface which offers a suitable rough surface for its attachments, where the film can remain undisturbed, may serve as a nucleus for its permanent retention. Incidentally, the suspended, very tenacious mucin has settled itself within the same region even before the pellicle is completely formed, and most likely it is primarily responsible for the initial starting-point for the calcareous deposit. The freshly precipitated flocculent sediments receive additions continuously from the uninterrupted slowly flowing saliva ; food debris, hæmoglobin, and other extraneous matter lend body and colour to the deposit and its own increase condenses its bulk. The initial focus for tartar formation is thereby created ; its future growth and physical characteristics are matters of time and circumstances.'

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[1] *Dental Cosmos*, 1921, 373.

(5) Composition.—Berzelius gives the composition of the calculus as follows :—

Phosphates of lime and magnesia	79·0
Salivary mucus	12·5
Ptyalin...	1·0
Animal matter soluble in HCl	7·5
	100·0

These figures can only be approximate. Stevenson gives the following analyses of the two varieties :—

	Water and organic matter.	Inorganic.
Soft tartar	21·48	78·52
Hard tartar	17·51	82·49

The deposit found on the buccal surfaces of the maxillary molars is composed almost entirely of calcium carbonate. The deposit on the lingual surfaces of the mandibular incisors contains much more calcium phosphate than carbonate, owing to the excess of the phosphates in the submaxillary secretion.

(6) Sequelæ.—As the presence of salivary calculus on the teeth gives rise to oral sepsis, particularly when it is present in any amount, it may give rise to any of the sequelæ which oral sepsis may cause. The variety of oral sepsis caused is that which is usually known as 'open sepsis,' so called because the discharge from inflamed and ulcerated surfaces may escape into the mouth and be swallowed with saliva or food ; even so, though the bulk of organisms so swallowed are at first destroyed by the acid of the gastric juice, it does not follow that their toxins are, and there is no doubt that some of these latter are absorbed locally into the circulation. The local effects it gives rise to are gingivitis and its sequelæ; these are brought about partly by the fact that stagnation areas are produced by the ledges of calculus overhanging the gingival margin. These overhanging ledges, by pressing back the gum, create a further area for the accumulation of calculus, so that a vicious circle is produced; these processes lead on to further recession of the gum accompanied by inflammation and the ultimate destructive effects of parodontal disease, so that the teeth are loosened and ultimately fall out. Accompanying these processes, local absorption of toxins and contact of the tongue with the roughened surface of the calculus may cause glossitis, particularly when portions of calculus break away. The lodgment of gross particles of food interdentally also occurs, and fœtor of the breath, often intense, usually occurs as well.

(7) Treatment.—The treatment of salivary calculus consists in thoroughly removing all the deposits and polishing the surfaces of the teeth. The force for detaching the deposit may be used in a direction towards the gum or away from it. Force is best used towards the gum when there is much tartar, and away from it when the quantity is small. When using force in a direction towards the gum, the right hand should be steadied by placing a finger or fingers upon the teeth, the cutting edge of the instrument being placed upon the tartar and a pushing motion used, when the tartar will be found to come off in large flakes. The greater part of the tartar can be removed in this way, the remaining adherent portions being removed in a direction from below upwards, the instrument being prevented from slipping by supporting the hand on the cutting surface of the teeth. At the completion of the scaling, the teeth should be polished with fine pumice-powder and soap applied by means of a circular brush on the dental engine.

(B) SERUMAL CALCULUS.

In dealing both with marginal gingivitis and parodontal disease reference was made to the frequent presence of **calculus under cover of the gum margin.** In character it is hard and dark, and is found more frequently on the approximal surface than on the labial and palatal surfaces. The source of this deposit is not clear, but it is highly probable that it is a deposition from the serous exudate and pus which occur round the necks of teeth in marginal gingivitis and in the pockets in parodontal disease. The deposit is found in the gingival trough on teeth where there is or has been a marginal gingivitis. The presence of the calculus is generally marked by a hyperæmic condition of the gum margin. The roots of teeth which have been the subject of parodontal disease are often covered with a calcareous deposit (see figs. 679 to 681). Analyses of these deposits show them to be composed mainly of phosphates. Oxalates are sometimes present. Its dark and sometimes greenish colour is probably due to the presence of altered blood pigment.

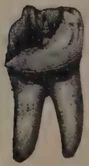

FIG. 679. — Showing well-marked deposit around the neck of a mandibular molar.

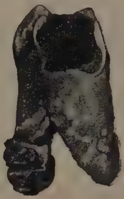

FIG. 680. — A maxillary molar showing large deposit of calculus on the apical portion of the palatine root.

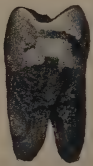

FIG. 681.—A maxillary molar showing irregular deposit of calculus over a large portion of the surface of the root.

F. C. Wilkinson is of opinion that serumal calculus consists of a mass of calcified epithelial cells.[1]

Occasionally calculus is present, and yet the gum tissues appear to be quite healthy, but in such cases it is probable that marginal gingivitis has occurred at an earlier period.

Treatment.—The deposit must be removed and the condition of the gum, or parodontal tissues, treated.

(C) STAINS.

Teeth may be discoloured by *extrinsic* or *intrinsic* stains, the former being deposited on the outside of the tooth and the latter staining its substance.

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[1] *Dental Record,* 1935, lv, 105.

Extrinsic Stains.[1]

(1) Green Stain.—A green stain is often found on the teeth, especially in the young. The colour varies from a greyish-green to bluish-green. It appears on the maxillary teeth more frequently than on the mandibular. The labial surfaces of the anterior maxillary teeth are the most frequent sites, the discoloration commencing at the gingival margin and encroaching on the surfaces in a direction towards the cutting margin. At times, more than half the surface is covered. The stain may extend to the approximal or even to the gingival surfaces. The stain may be limited to any pits, grooves, or depressions present on the surface of the tooth. The green stain is nearly always a sign of lack of cleanliness on the part of the patient. The stain is intimately connected with the enamel cuticle. Removal of the stain by acids leaves a colourless surface behind. Green stain may occur beyond the enamel margin on the teeth of adults.

Cause.—The origin of green stain is not clear. The following facts are known: The colouring matter is characterised by its insolubility in nearly all the ordinary solvents, viz. glycerine, alcohol, chloroform, turpentine, etc.; tincture of iodine has no decolorising effect. The colouring matter is rapidly destroyed by chlorine and hydrogen peroxide. Calcination experiments show that the green stain deposit may be either of an organic or inorganic nature. There is reason to believe from experimental investigations that in certain cases the green colour may be due to the presence of sulpho-methæmoglobin. Recent investigation by Pincus[2] suggests that the staining is caused by the pigment pyocyanin (oxymethylphenazine $C_{26}H_{20}N_4O_2$), a phenazine derivative formed by the *Bacillus pyocyaneus*, an organism which may be found in these mouths.

When the stain is removed the enamel appears quite normal in the majority of cases, but in some cases the surface shows signs of decalcification.

Treatment.—The stain should be removed by means of suitable wheels or brushes on the dental engine, a little powdered pumice stone being used to increase the abrasive action. Acid application should *not* be used.

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[1] The matter contained in the following section relating to extrinsic 'stains' has been obtained almost entirely from a paper by Miller (*Dental Cosmos*, April 1894), entitled 'The Deposits upon the Teeth with Special Reference to Green and Metallic Deposits.'

[2] *Brit. Dent. Journ.*, 1941, lxx, 52.

(2) Metallic Stains.—(*a*) *Copper.*—The teeth of workers in copper invariably show a green stain. Miller examined 150 persons who had been working in copper for more than a year and found distinct discoloration of the teeth in all of them. The colour varies from green to dark dirty green, reddish-green, bluish-green, greenish-blue to bluish-purple, the latter being seen in ‘ phosphor-bronze ’ workers. Persons using brass musical instruments at times show staining of the teeth.

The gingival margin of copper workers is hyperæmic, owing to irritation from the more or less oxidised particles of the metal which may have worked their way up into the gums.

(*b*) *Iron.*—Iron seems to produce a brown stain of varying tints. In workers in iron, Miller found that nearly all showed brown spots or deposits on their teeth, the mandibular teeth being more frequently discoloured than the maxillary. Medicines containing iron may stain the teeth if the mouth is not cleansed each time after taking the medicine. The forms of iron detrimental to the teeth are those containing free acid, such as the perchloride.

(*c*) *Mercury.*—Deposits of mercury in the form of sulphide are seen on teeth filled with amalgam. These deposits occur also on the teeth of patients subjected to prolonged mercurial treatment, the mercury affecting the teeth through the saliva. The stain may be due to the use of mouth-washes containing mercury. The presence of the mercury on the teeth can be demonstrated by chemical tests.

(*d*) *Lead.*—Lead poisoning may produce a blue line in the gum margins (p. 510). According to Hirt,[1] in chronic lead poisoning the teeth are discoloured and appear dark brown at the necks, shading to light brown towards the occluding surfaces.

(*e*) *Nickel.*—A tooth from the mouth of a worker in nickel is figured by Miller. The deposit is thick and of an opaque bluish to greenish-black colour.

(*f*) *Silver.*—Silver, when used in the mouth, produces a dark stain, which is due to the formation of the sulphide.

Treatment of Extrinsic Stains.—As the above stains are superficial deposits and not intrinsic stains, they can be removed by the use of abrasives.

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[1] Quoted by Miller.

Intrinsic Stains.

Besides those mentioned on page 39, there is a variety of intrinsic stains which may cause discoloration of teeth.

The most important of these are those *stains which are derived from the blood pigment* and which cause discoloration of the tooth by finding their way along the dentinal tubes after death of the tooth pulp ; such staining is especially seen in the anterior teeth of the young when there has been an extravasation of blood from traumatic injury of the tooth pulp, causing its death ; the dentinal tubes being patent at this age, staining reagents very readily penetrate the dentine, sometimes within a few hours, whereas in a middle-aged or elderly person the tubes are not so patent and penetration by stain does not take place so readily or may not take place at all. If a tooth pulp has decomposed, the chemical changes in it may give rise to iron compounds, derived from blood pigment, which cause the discoloration. In addition, there are a number of metallic stains.

Copper amalgam, when inserted into a moist cavity, or one from which the whole of the carious dentine has not been removed, or if the amalgam has not been cleaned in dilute acid and dried before insertion, will stain teeth a greenish colour. Even the ordinary *silver-tin-zinc alloys*, though not usually staining teeth, may, in sulphur spring districts such as occur in parts of New Zealand, stain the teeth black by the production of silver sulphide.

Other metallic stains also used to occur, when metals not now used in amalgams were employed in conservative dentistry.

Fatty substances, formed in some degenerations of the tooth pulp, may also cause discoloration.

Similarly *some of the drugs* used on teeth, notably the essential oils, may cause discoloration, but such drugs as iodine, silver nitrate, and potassium permanganate should not be allowed to come into contact with newly exposed dentine, particularly in the young, if discoloration is to be avoided.

Treatment of Intrinsic Stains.—Treatment consists in attempts to remove the discoloration by producing new and soluble or colourless metallic salts by chemical means and washing them away, or by dissolving them out in media in which they are soluble, and is generally referred to as bleaching.

Before any attempt is made to bleach teeth intrinsically stained all carious dentine must be removed and the pulp canal treated, and this, of course, applies to the periapical area if it is infected. No bleaching agent should be used or sealed in till the greater portion of the pulp canal has been efficiently sealed, lest the chemicals used in the process leak through the apical foramina, where they could cause considerable damage.

The bleaching agents which may be used are *oxidising agents*, *sulphur dioxide*, *chlorine*, and *iodine*; the two latter are of especial value for the conversion of metallic deposits into their soluble salts.

Oxygen may be applied in the nascent state by the use of peroxide of hydrogen in any strength; it is of particular value in stains derived from blood pigment. Hydrogen peroxide has usually a slightly acid reaction due to impurities, so after its use it is wise to neutralise the area treated with a weak solution of bicarbonate of soda, which also helps to remove any fatty or oily substances which may be present.

Sodium peroxide may also be used for this purpose in the form of powder; metal instruments should not be used to convey it. When it is in place it should be moistened; it then gives off its oxygen with some heat, and indeed care is needed in its use as it may act with almost explosive violence. In the reaction sodium hydroxide is produced, which can combine with fatty and oily substances to form soluble soaps.

Sulphur dioxide is produced *in situ* by the action of boracic acid on sodium sulphite; a mixture of the powdered drugs is moistened *in situ* and sulphur dioxide is set free. It acts by its conversion when in solution from sulphurous to sulphuric acid, so the area should be neutralised afterwards by the use of a dilute solution of sodium bicarbonate.

Chlorine is produced by the action of acetic or any other acid on bleaching powder (chlorinated lime); the powder may be placed *in situ* and there moistened with the acid solution.

Iodine may be applied as the tincture of iodine to act on metallic salts for the production of soluble iodides; such iodine stain as remains may be removed by the action of sodium thiosulphate, when colourless sodium tetrathionate is produced and washed away.

These reagents need to be applied for some time and at several sittings, and may, when possible, be sealed in for several days to prolong and intensify their action. The results are usually a considerable improvement in the condition, though often not extending to perfection, but treatment can often be aided by the removal of some of the stained tissue (dentine), though enough must be left to leave sufficient support for the remaining enamel. The result is greatly helped by a final thorough dehydration of the treated tissue with absolute alcohol and hot air, and the immediate sealing up of the exposed dentinal tubes by oxyphosphate of zinc cement or copal varnish before the final restoration is completed.

CHAPTER XXI

Odontomata and other Abnormal Productions derived from Dental Formative Organs.

CLASSIFICATION—THE EPITHELIAL PRODUCTIONS.

The term 'odontôme' was first used by Paul Broca,[1] in Paris, on December 30, 1867.

The use of the word 'odontoma' (or odontome) should be confined to neoplasms derived from dental formative organs; and a neoplasm is best defined as *autonomous new growth*, such growth being unco-ordinated with the normal.

The only abnormal productions of dental formative organs which conform to these data, wholly or partly, are: some complex odontomes (and their multiples, *i.e.* compound odontomes), dilated odontomes, such basal-celled carcinomata (multilocular cysts) as may be of dental origin, and possibly some fibrous odontomes and cementomata.

An odontome therefore may be defined as *a neoplastic production derived from dental formative organs.*

In the remainder of these formations there is no evidence of neoplastic growth, and they could well be classified according to their etiological origin as due to trauma, infection, and congenital defect or variation—this last being occasionally hereditary; and such classification has much to commend it. The fact that their co-ordination of growth is never wholly absent and sometimes perfect, definitely bars them from classification as neoplasms, and hence as odontomes.

At the same time we know that trauma [2] and certain chemicals may bring about unco-ordinated growth and occasionally neoplastic growth,[3] and the same may be true of bacteria and their toxins ; we also know that partly unco-ordinated growth of dental tissues has occurred in different members of the same family,[1] and that it has occurred symmetrically,[2] so it is not always clear into which category any particular specimen should be placed, even when its accurate history is available.

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[1] Paul Broca, *Gaz. heb. de Méd. et de Chir.*, Paris, 1868, Ser. 2, 5, 19, 70, 113.

[2] Sir Frank Colyer, 'Variations and Diseases of the Teeth of Animals,' London, 1936, 722.

[3] (*a*) E. L. Kennaway, *Journ. Path. and Bact.*, 1924, 27, 233.
(*b*) *Idem.*, *Brit. Med. Journ.*, 1925, 2, 1.
(*c*) *Idem.*, *Biochem. Journ.*, 1903, 24, 497.
(*d*) J. W. Cook, I. Hieger, E. L. Kennaway, and W. V. Mayneord, *Proc. Roy. Soc.*, Ser. B, 1932, 111, 455.
(*e*) J. W. Cook, *ibid.*, Ser. B, 1932, 111, 485.
(*f*) G. Barry, J. W. Cook, G. A. D. Haslewood, C. L. Hewett, I. Hieger, and E. L. Kennaway, *ibid.*, Ser. B, 1935, 117, 318.

On these accounts it is more instructive to the student to group all abnormal productions derived from the dental formative organs together, as when classified in this manner their relationship to each other from the evolutionary point of view is more readily appreciated ; it must, however, be emphasised that *the term odontoma used in its correct sense embraces only those abnormal productions which are neoplastic in origin*, whether they ultimately give rise to calcified dental tissues or not, and the term should not be loosely used.

Thus some productions due to trauma and infection (see figs. 788 to 793) may very much resemble true odontomata and possibly be differentiated only by their accurate history being known.

Similarly cysts (apart from cyst-adenomata) cannot be regarded as coming within the definition of surgical tumours, and so should not be classed as odontomata.

The whole of these productions may be primarily classified according to the tissue or tissues from which they have origin as :—

(A) Epithelial.
(B) Composite (epithelial and mesoblastic).
(C) Connective tissue (mesoblastic).

(A) **Epithelial Productions.**—Where the abnormal development takes place in dental epithelium alone :—

(1) Cysts of eruption.
(2) Dental cysts.
(3) Dentigerous cysts.
(4) Multilocular cysts (possibly a small percentage only have origin from the dental formative organs).

(B) **Composite Productions.**—Where the abnormal development takes place primarily in dental epithelium and secondarily in mesoblastic dental tissues.

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[1] Schmitz, and A. Witzel, *Deuts. Monats. f. Zahn.*, 1901, 19, 126.

[2] M. Oudet. See report by MM. Hipp. Cloquet and Duval in *Bull. de la Fac. de Méd.*, Paris, 1821, 7, 369. Also cited in *Nouveau Journ. de Méd.*, Paris, 1821, 10, 245.

Putting the simpler forms first, these may be classified as follows :—

(1.) Arising from Tooth Germs of the Normal Series.

(a) ADICHOTOMOUS ABNORMALITIES. These are imperfectly co-ordinated teeth outside the range of normal variation; and some partly unco-ordinated forms: Some dilated odontomes.

(b) INCOMPLETE DICHOTOMIES.
- i. Co-ordinated (resembling the normal)
  - Of part of the tooth germ: Extra cusps and roots.
  - Of the whole tooth germ: Incomplete equal dichotomies.
- ii. Imperfectly co-ordinated (part or the whole unlike the normal).
  - Of part of the tooth germ: Enamel nodules.
  - Of the whole tooth germ: Incomplete unequal dichotomies.
- iii. Partly unco-ordinated: Some dilated and some complex odontomes.
- iv. Unco-ordinated: Most complex odontomes (called compound[1] when multiple).

(2.) Arising from Tooth Germs in excess of the Normal Series. These are either complete dichotomies of germs of the normal series, or arise *de novo* from the dental lamina; they are all supernumerary formations.

COMPLETE DICHOTOMIES.
- i. Co-ordinated (like the normal): Complete equal dichotomies.
- ii. Imperfectly co-ordinated (unlike the normal): Complete unequal dichotomies.
- iii. Partly unco-ordinated: Some dilated and some complex odontomes.
- iv. Unco-ordinated: Most complex odontomes (called compound[1] when multiple).

(C) **Connective Tissue Productions.**—Where the abnormal development takes place in mesoblastic dental tissues alone :—

(1) Fibrous odontomes.
(2) Cementomes and cemental hyperplasia.

Dental formative organs giving rise to calcified dental tissues in varying stages of co-ordination of growth may also occur in teratomata in the ovary, testis, pituitary and pineal glands.

(A) EPITHELIAL PRODUCTIONS.

(1) Cysts of Eruption.—Small cysts are occasionally seen over teeth which are due shortly to erupt or whose eruption is overdue; usually they are so small as not to interfere with eruption. Occasionally they reach an abnormal size; when this occurs the overlying tissues become thinned and they present a bluish appearance and fluctuation may be felt. They occur over both deciduous and permanent teeth, and if large may interfere with eruption, and this they occasionally do when formed in connection with mandibular third molars, the eruption of which from one cause or another is often delayed ; sometimes their growth over this tooth is such that they tend to surround it, or the tooth tends to erupt into it, so that in this manner a form of dentigerous cyst is produced (fig. 703).

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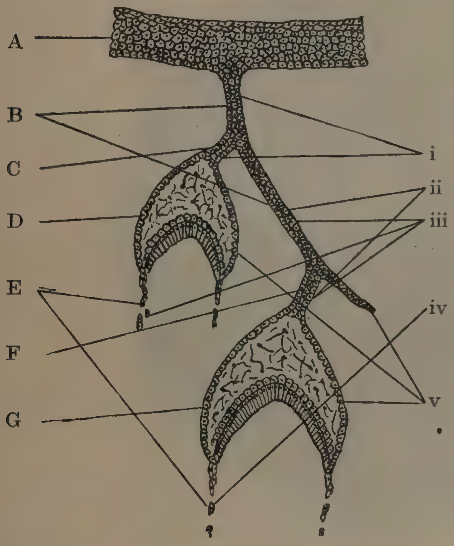
[1] The formations of compound odontomes may be of any or all types, but are rarely of good co-ordination.

Histologically, they are lined with 'epithelium many layers thick, spheroidal at the growing periphery, and tending to become stellate on the cyst side.' (J. G. Turner.)

Origin.—It is simpler to consider the origin of such cysts when they overlie deciduous teeth or those permanent teeth which have no predecessors ; the possibly more extensive origin in the case of those teeth which have deciduous predecessors is apparent in the diagram (fig. 682). In the former case the only epithelial cells from which they can arise are those remnants of the tooth band which lie superficial to the tooth.



Site of cells which probably give rise to :—

- i. Cyst of eruption over a deciduous tooth. Dentigerous cyst enclosing a deciduous tooth.
- ii. Cyst of eruption over a permanent tooth which has no predecessor. Dentigerous cyst enclosing a permanent tooth which has no predecessor.
- iii. Dental cyst on a deciduous tooth. Dentigerous cyst enclosing a permanent tooth which has a deciduous predecessor (commonly).

(ii and iii) Cyst of eruption over any permanent tooth.

- iv. Dental cyst on a permanent tooth (or dentigerous cyst when it has tracked and enclosed a third molar).
- v. Multilocular cyst (possibly a few only).

FIG. 682.—Diagram of named parts of epithelial dental formative organs. (This diagram is not true of any age.) A, surface epithelium ; B, tooth band ; C, neck of deciduous enamel organ ; D, deciduous enamel organ ; E, sheath of Hertwig ; F, neck of permanent enamel organ ; G, permanent enamel organ. (E. S. del. et fec.)

Normally, these epithelial remnants of the tooth band constitute the epithelial portion of the gubernaculum.

Etiology.—Little is known of the stimulus which causes these epithelial remnants to proliferate. It is believed that normally these cells degenerate and so open up the path for the eruption of the tooth, so that their proliferation must be looked on as a perversion of the normal process. Infection is frequently absent when these cysts occur ; it is possible that the unduly prolonged stimulus of a tooth slowly erupting or unable to erupt may bring about such perversion of growth. It occasionally occurs in connection with unerupted supernumerary teeth, so giving rise to dentigerous cysts ; and with compound composite odontomes, when the supernumerary formations before eruption may be involved in cysts, some or all of which may ultimately coalesce.

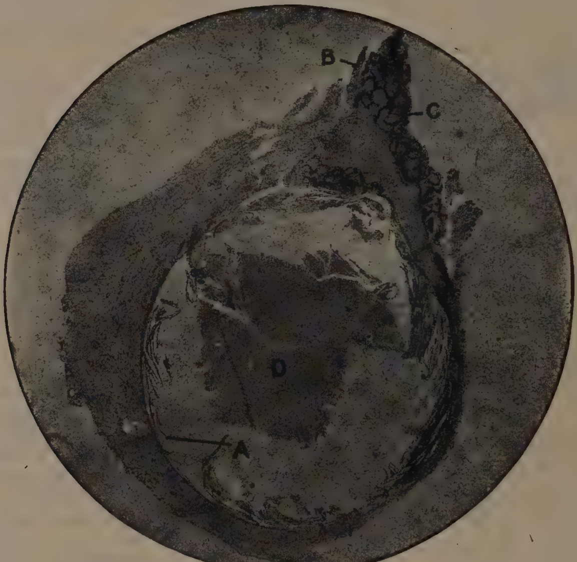


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Fig. 683.—Section of a small cyst attached to the apex of a root. A, epithelial lining ; B, mass of epithelium ; C, remains of epithelial reticulum ; D, semi-solid contents. (Turner.)

Pathology.—The cells commence to proliferate, the epithelial mass is non-vascular, being nourished only by surrounding lymph ; as proliferation increases the central cells therefore become starved, undergo hydropic degeneration, become disorganised, and so give rise to the serous fluid found in these cysts.

If a tooth erupts into the cavity of such a cyst or is enveloped by it in its growth, a variety of dentigerous cyst is produced, but such is not the usual way in which dentigerous cysts arise, though it is probably the way in which, in the absence of infection, dentigerous cysts involving deciduous teeth and most third molars arise, particularly when the latter have been retained in the jaws unerupted for many years.

Treatment.—If they are so small as not to cause any interference with eruption, simple puncturing to allow the escape of the serous contents of the cyst will suffice, but if they are of such size as to interfere with the eruption of the tooth it is necessary to remove the tissue overlying the tooth.

(2) **Dental Cysts.**—Dental cysts as typically seen are epithelial lined cysts which occur in the jaws (fig. 683) and are usually formed directly in connection with a tooth having a dead pulp and periapical infection. They may occur at any age and in connection with any tooth; one of us has seen one at the age of seventy-six; they are commonest from the twentieth to the fortieth year. They occur more commonly in connection with the permanent teeth, and in 1922 [1] and subsequently have been found in connection with deciduous teeth (figs. 684 to 686). Their occurrence in connection with deciduous teeth is not so common because these teeth are present for so short a time.

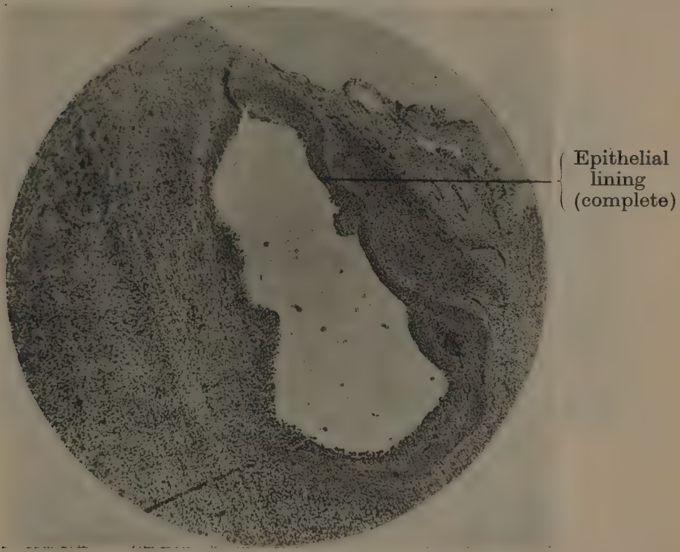

FIG. 684.—Photomicrograph of small entire cyst *in situ* attached to part of a deciduous tooth. (E. S.) (From *Proc. Roy. Soc. Med.*)

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), 1922, 15, 56, and 1927, 20, 67. (E. S.)

They give rise to slowly increasing smooth, globular, painless swellings which cause the absorption of adjacent bone and

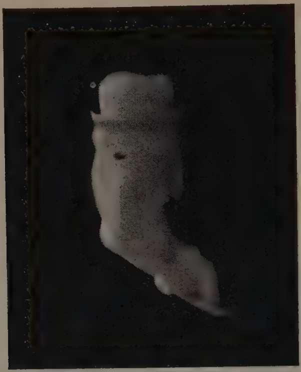

Fig. 685.—A deciduous tooth with a portion of a cyst wall attached. (E. S.) (From *Proc. Roy. Soc. Med.*) (Now in odontological collection at the Royal College of Surgeons of England.)

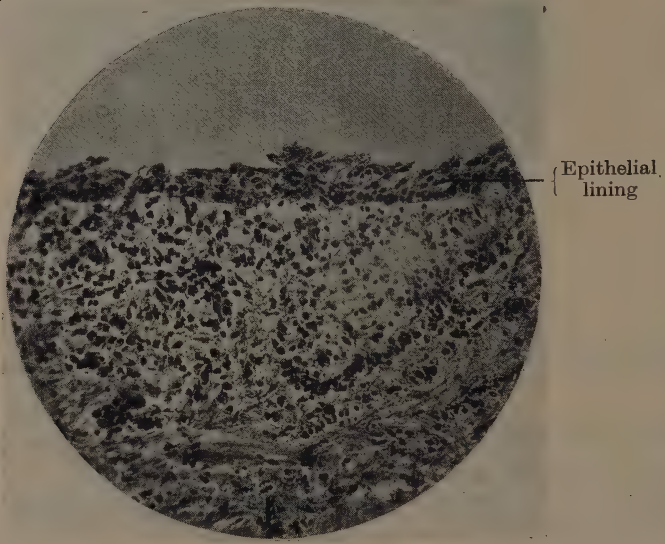

Fig. 686.—Photomicrograph of portion of cyst wall of fig. 685. (E. S.) (From *Proc. Roy. Soc. Med.*)

increase in the direction of least resistance. While the adjacent bone is being absorbed there is a deposition of new bone externally, but the rate of deposition is not so fast as that of absorption, so that at the stage when the bone is so thin that it may be dented on pressure a typical crepitus may be elicited. As the size increases the bone disappears and fluctuation may then be obtained, the fluctuating area being frequently bounded by a bony edge. There is increased tension within the cyst. There are usually no inflammatory symptoms. Occasionally they become infected and suppurate and then simulate chronic abscesses, with the difference that the epithelial lining still remains *in situ*. Not infrequently they burrow along the medullary portion of the bone below the apices of teeth, and may then stimulate the rests of Malassez of neighbouring normal teeth to proliferate and, coalescing with the new cyst caused in this manner, produce loculation (see fig. 698). Radiograms usually show a sharp outline to the cyst (in contrast to the more blurred outline of a chronic abscess) (figs. 687 and 688). They usually occur singly, but multiple cysts have been recorded. If a dental cyst occurs in connection with a deciduous tooth it may in its growth envelop the successional permanent tooth and so become a dentigerous cyst (fig. 689). Similarly, dental cysts formed in connection with a mandibular first permanent molar may burrow along the cancellous portion of the bone and envelop the unerupted third molar. The removal of the tooth they are formed in connection with usually opens them, and sometimes, if not too large, they come away entire and attached to it ; but sometimes they remain unruptured in the jaws and continue to increase in size. The outer alveolar plate being the thinner,

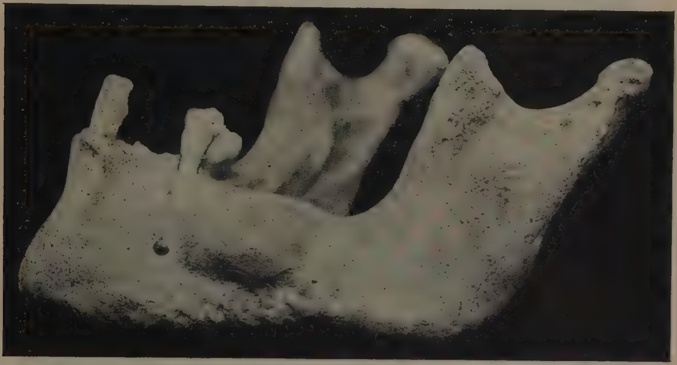

FIG. 687.—Mandible with a cyst in the molar region.

their increase in size nearly always causes the swelling to be first noticed on that aspect (fig. 687). If they arise in connection with maxillary teeth, the apices of which are in relation to the antral floor, the antral lining is pushed up and displaced by the cyst so that the air cavity is obliterated (figs. 690 to 693).

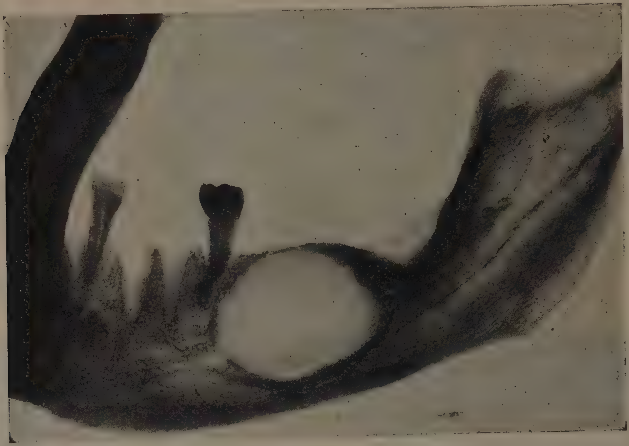

Fig. 688.—Radiogram of the specimen shown in fig. 687.

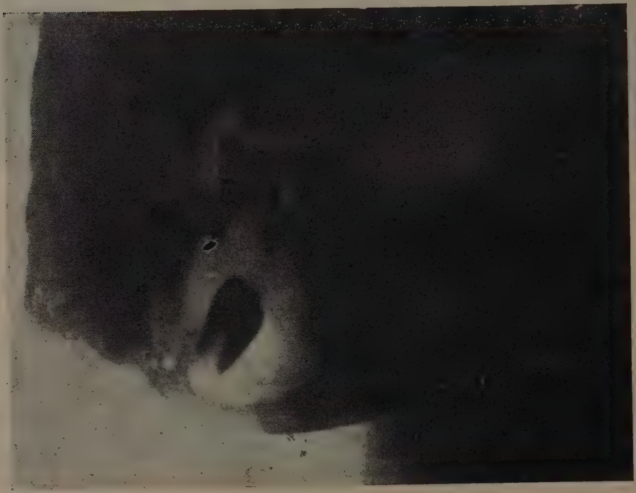

Fig. 689.—Dental cyst formed in connection with a deciduous tooth enveloping a premolar. (E. S.) (From *Proc. Roy. Soc. Med.*)

Histologically, they consist from without inwards of a layer of thinned-out bone with osteoclasts on its inner aspect (dependent on the size of the cyst, this layer may not be present), a layer of fibro-connective tissue with its fibres arrayed concentrically forming the capsule, which may be very thick, and a layer of epithelium (figs. 694 and 695). The layer of epithelium varies

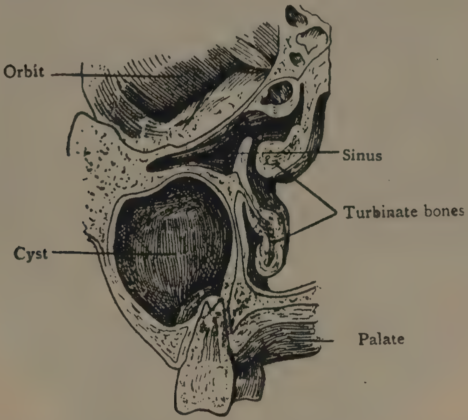

FIG. 690.—Dental cyst pushing up the sinus until it is a mere slit beneath the orbit. Modified from Zuckerkandl. (From *Brit. Dent. Journ.*)

FIG. 691.—Cyst originating in connection with a maxillary molar.

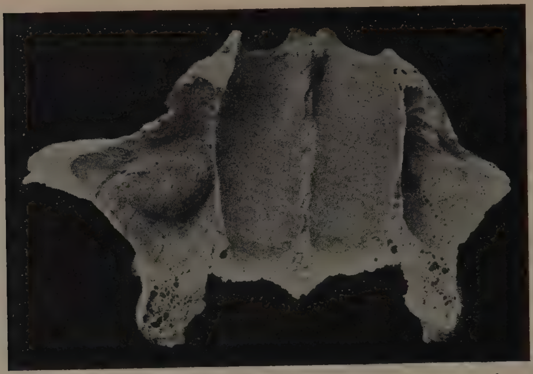

FIG. 692.—Section through the maxillary sinuses of the specimen shown in fig. 691. The cyst has pushed upwards into the sinus.

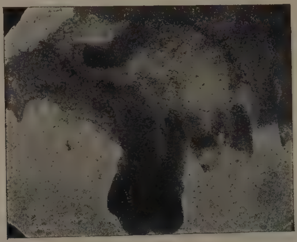

FIG. 693.—Radiogram of the specimen shown in fig. 691.

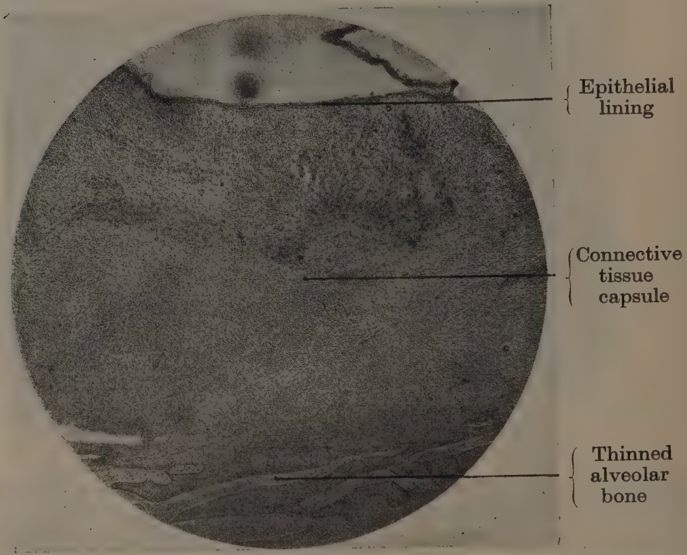

FIG. 694.—Section of a portion of cyst wall. (E. S.) (From *Proc. Roy. Soc. Med.*)

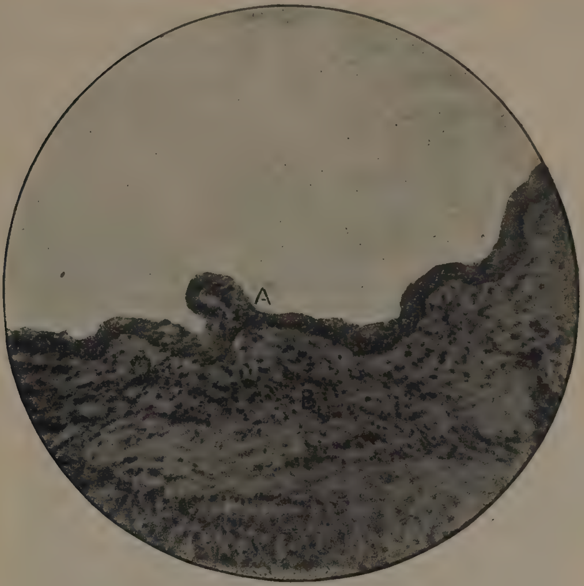

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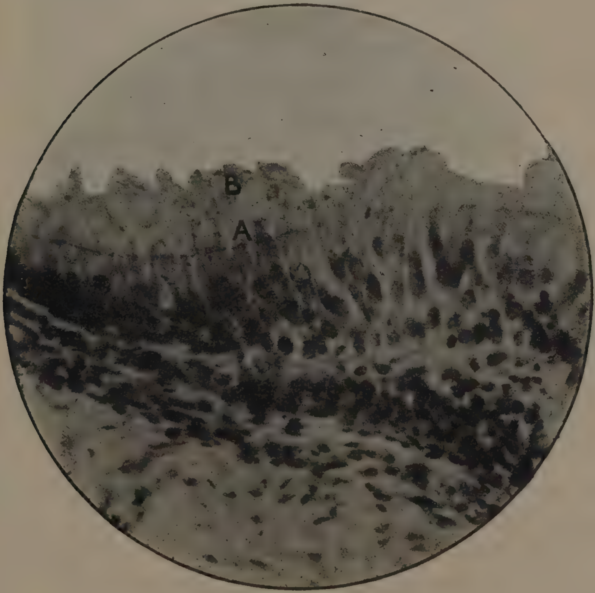
FIG. 695.—Section of wall of large dental cyst. A, thin regular lining of epithelium ; B, connective tissue capsule. (Turner.)



× 350, ⅛-in. obj., N.A. 0·6.

FIG. 696.—Section of dental cyst showing at A, ciliated epithelium ; B, products of degeneration or secretion. (Turner.)

considerably; in large cysts it may be so thinned out as to be only one or two cells deep or even absent in extensive parts, in small ones it may be up to forty cells in depth and present interpapillary processes on its outer aspect which occasionally loop over and join other similar processes. Stratified epithelium is sometimes present and sometimes, particularly in small cysts in young people, it is of a more columnar type, and on several occasions ciliated epithelium has been described (fig. 696). The cells immediately bordering the cyst cavity are in a state of

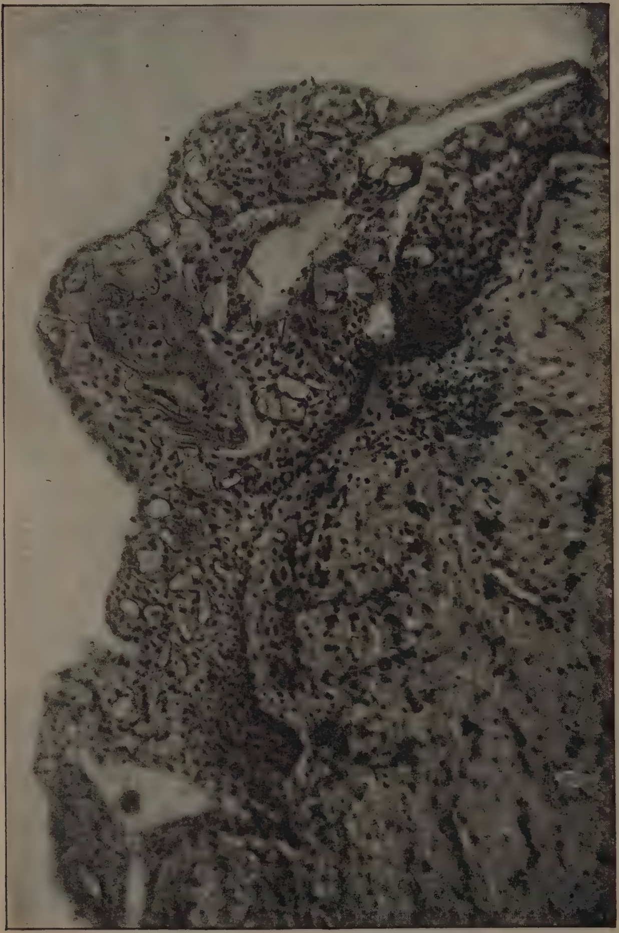


FIG. 697.—Wall of a dental cyst angiomatous in structure, and with thick epithelium, in a state of marked hyaline degeneration. (*Dental Cosmos*, July 1918.)

degeneration and sometimes show stellate forms. Fat globules have been described as constantly present.

The walls of cysts occasionally show an angiomatous structure (fig. 697). The contents are usually a yellowish or brownish, translucent, mucus-like fluid of varying viscidity, with a varying amount of crystals of cholesterin present; these latter cause the contents to glisten, the glistening varying according to the amount of cholesterin present. Proteins in the form of serum albumin and serum globulin are present, the latter being more abundant, and also a small quantity of nucleo albumin. Occasionally the contents are a semi-solid whitish mass resembling cerebral matter and feeling greasy to the touch; and E. B. Dowsett [1] records a case where the cyst contained a hard substance of leathery consistency, dead white in colour, which microscopical examination showed to be layer upon layer of keratinised epithelium devoid of vessels.

A case has been recorded by A. T. Pitts where, after opening a cyst, a hair was found growing from the epithelial lining; it must be remembered, however, that abnormal epithelium may be capable of metaplasic changes and so capable of taking on the potentialities of other forms of epithelial cells; this may also explain the occasional incidence of ciliated epithelium lining these cysts.

It is said that in all cases micro-organisms are present in the cyst wall and sometimes in the contents, though these often appear sterile.

Origin.—It has been shown by J. G. Turner [2] that dental cysts formed in connection with permanent teeth have their origin from epithelial cells left in the periodontal membrane and periapical tissues; these cells, which are remnants of the epithelial dental formative organs, are the 'rests' of Malassez which, in the active period of their life, made up the epithelial sheath of Hertwig (fig. 697A). When they occur in connection with deciduous teeth there are other possible sources of origin besides the rests of Malassez; the cells of all these possible sources are, however, very crowded together and the actual source is probably not of much importance (see fig. 682).

Warwick James and McIntosh, quoting three cases, suggest that the origin of the epithelium may be a downgrowth from the

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[1] *Proc. Roy. Soc. Med.*, 1933, xxvi, 1562.

[2] *Brit. Dent. Journ.*, 1898, xix, 711.

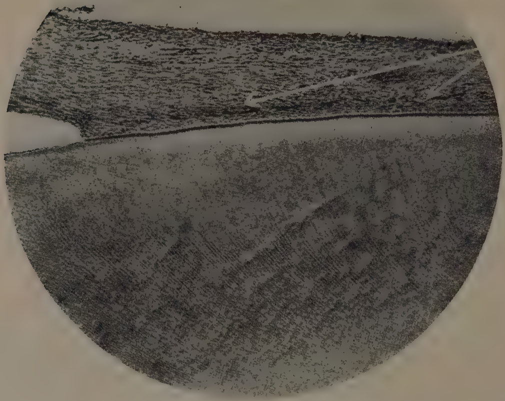

FIG. 697A.—Section showing 'rests' of Malassez as in a tooth about to erupt. (E. S.) (From *Brit. Dent. Journ.*)

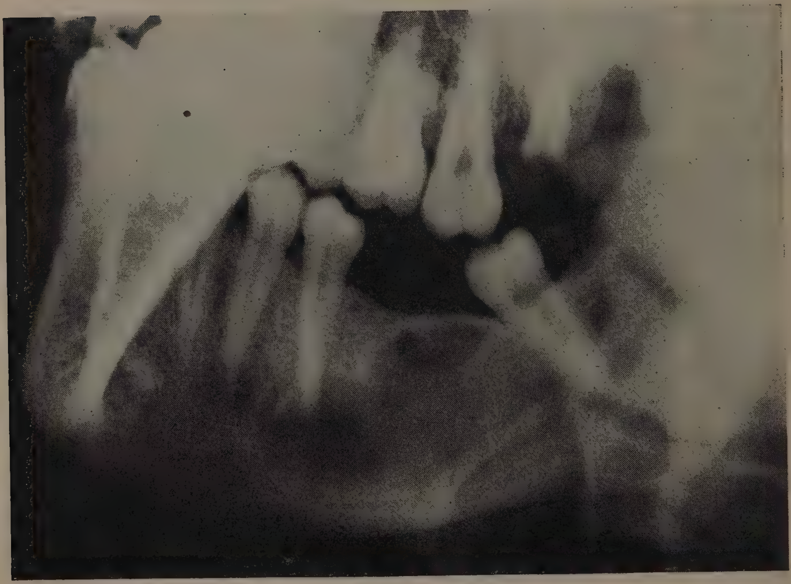

FIG. 698.—Large dental cyst of mandible originating from second molar root with extension under second premolar with living pulp and showing a loculated appearance. (E. B. Dowsett.) (From *Proc. Roy. Soc. Med.*)

gum along a sinus formed in connection with a pulpless and infected tooth, but it must be admitted that such sinuses are very rarely clinically observed; and the suggestion is somewhat contra-indicated by E. B. Dowsett,[1] who shows (fig. 698) that when a cyst has extended to the apical area of an adjacent tooth or teeth, even when they possess live pulps, a similar epithelial growth may be set up there, which, coalescing with the original cyst, may give it a loculated appearance. (See p. 689.)

Etiology.—The common causative factor stimulating these epithelial remnants into proliferative activity is derived from micro-organisms and/or their toxins in the pulp canals of pulpless and infected teeth; these find their way into the periapical or parodontal tissues and act as irritants. At first local reaction produces a mass of granulation tissue, but apparently the proliferation of the epithelial remnants found there occurs almost simultaneously, and the so-called epithelial granuloma is produced. (See Chapter XV.)

Apparently all such epithelial proliferations do not proceed to cyst formation.

It is probable that toxic or micro-organismal irritation in a parodontal pocket may start the proliferation of these epithelial cells, or even such trauma as may be occasioned by too rapid orthodontic movements, but histologically one cannot regard the latter as proved.

Pathology.—The epithelial cells having started to proliferate and being avascular and nourished only by surrounding lymph, increase in number till the central cells become so far removed from their source of nourishment that they become starved; they then undergo hydropic degeneration and become disorganised and give rise to the contents found in these cysts. The fibrous capsule is probably mainly made up by the fibres of the surrounding fibro-connective tissue arranging themselves concentrically in the direction of least resistance.

The differential diagnosis from other swellings of the jaw is given in Chapter XXXIII.

Treatment.—As a preliminary, the mouth should be rendered as clean as possible by the removal of calculus and the extraction of all septic teeth except the one in immediate connection with the cyst, or, if the cyst is a large one, those in close relation to it. When these wounds have healed the causative tooth should be removed and a large opening made into the cyst, usually by removing as large a portion of the outer wall as one conveniently can together with the epithelial lining immediately beneath it, so that the cyst cavity merges into the mouth cavity; the remaining epithelial lining may be left *in situ* so that it only remains for the edges of the wound to epithelialise over for surface healing to be complete. The contraction of surrounding connective tissue will in time obliterate the major portion of the cavity caused by the growth of the cyst.

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[1] *Proc. Roy. Soc. Med.*, 1931, xxv, 47.

Packing will only be necessary at the conclusion of the operation to arrest hæmorrhage, and is often not necessary even then. Frequent lavage is desirable so that discharge or debris may not collect in the cavity.

Circumstances may not always allow of as large an opening as is desirable being possible, particularly in the case of large maxillary cysts, and in these cases in particular (but in all others as well) it is most important to keep the opening widely patent and prevent its closure by scar contraction. For this purpose it is sometimes necessary, particularly in the case of maxillary cysts, that a vulcanite plug be worn to keep the opening patent.

Some dental surgeons prefer to conserve the epithelium covering the oral aspect of the cyst, raising it as a broad-based flap attached externally in the sulcus, and after removing a proportionate amount of the cyst lining, stitch this flap down in its place after the removal of any cyst lining from its deep aspect.

Though the operative treatments as described above have been practised for a number of years with great success *there is a rare but occasional risk in not removing the whole of the epithelial lining from a cyst* (see p. 714). It consists in the possibility of neoplastic growth taking place in an unremoved and possibly hidden part of the lining and has been known personally and from records to have occurred on at least eight occasions. As it is axiomatic that avoidable risks should not be taken it is wiser to revert to the older operation of removing the whole of the lining unless special circumstances should indicate otherwise. In any case small cysts are always better removed completely, and the wound allowed to heal either by granulation or under a flap raised to facilitate access.

In the process of healing after operation the deposition of new bone in the mandible soon restores it to its normal contour, and though new bone is not so readily or extensively formed in the maxilla, E. B. Dowsett[1] has clearly demonstrated that a considerable portion of the bone lost by pressure absorption in the case of maxillary cysts may be replaced there also.

(3) **Dentigerous Cysts.**—These are cysts which usually occur before the age of twenty years, being commonest about the fifteenth year; their name implies that they contain a tooth or part of one, or it may be more than one tooth. Any tooth may be contained in the cyst, but, clinically, premolars and permanent canines are most often involved, though third molars and maxillary incisors are by no means rare (figs. 699 to 701). The occurrence of deciduous teeth[2] and permanent first molars in cysts is very rare.

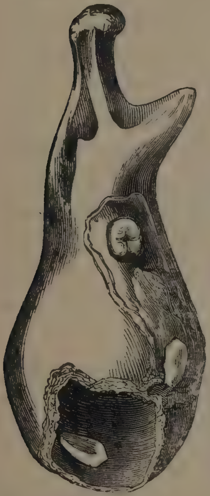

Fig. 699.—Portion of a mandible showing a large dentigerous cyst.

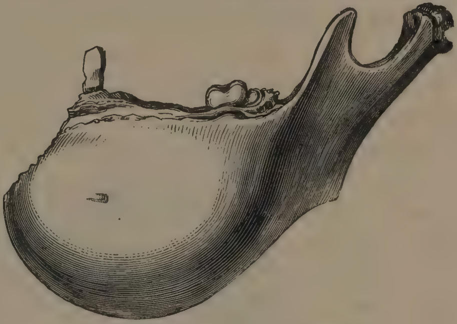

Fig. 700.—Same as fig. 699.

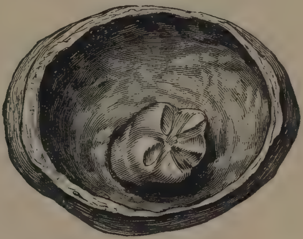

Fig. 701.—A dentigerous cyst (natural size). (From *Trans. Odonto. Soc.*, xxvi, 182.)

The clinical appearances are nearly always identical with those of dental cysts save that the tooth involved in the cyst is absent from its normal position in the mouth, but in some cases the age for its appearance may not have been reached. As most of them arise primarily as dental cysts derived from deciduous teeth, the causative pulpless and infected deciduous tooth may be present (fig. 689), but even when they do so arise the causative tooth is not always present. Apart from the above, their clinical history and appearance, signs, symptoms, and mode of extension are those of dental cysts.

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[1] *Proc. Roy. Soc. Med.*, 1931, xxv, 47.

[2] There are only two recorded cases of deciduous teeth of the normal series being found in cysts: that in Salter's 'Dental Pathology and Surgery,' 1874, 219, and that in Thoma's 'Clinical Pathology of the Jaws,' 1934, 280.

A radiogram shows a tooth or part of one, or teeth, within the cyst cavity. Teeth in excess of the normal number have been found in cysts, to the extent of 4 per cent. of all dentigerous cysts, both in this country and in the United States. They usually occur singly, but multiple cysts [1] have several times been recorded, sometimes symmetrically, and in a recently recorded case [2] of a family of seven children, four of them had no less than ten dentigerous cysts among them though they occurred singly in two of these four ; the parents were normal. Similarly, cystic formation often takes place around the multiple calcified formations of compound composite odontomes, and some or all of these cysts may coalesce.

Histologically, the cyst wall and contents are identical with those of dental cysts, except that the epithelium lining the cyst, being younger, does not so often occur in the stratified form and that there is a tooth or part of one enclosed in the cyst. Nasmyth's membrane is present on the teeth in cysts. Calcareous material has been described by Salter in the wall of one of these cysts (fig. 702).

Origin.—By analogy with dental cysts they originate from the parodontal epithelial remnants of Malassez formed in connection with deciduous teeth, but that there are other possible origins (but all from epithelial dental formative organs) may be gathered from fig. 682 and from the text concerning cysts of eruption and dental cysts.

Etiology.—When these cysts contain premolars, permanent canines, or incisors, they are nearly always dental cysts formed in connection with pulpless and infected deciduous teeth which have, in their extension, enveloped their successional tooth ; apart from the fact that they contain a tooth their etiology has therefore been discussed under dental cysts.

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), 1922, 15, 56. (E. S.)

[2] *Journ. Amer. Dent. Assoc.*, 1938, xxv, 623. (See also : *Brit. Journ. Radiol.*, 1938, ii, 227, for an account of another case of familial multiple dentigerous cysts.)

Should they contain third molars, which is not uncommon, the tooth while unerupted may have become enveloped by the cyst (possibly having origin from a permanent first molar, where they commonly occur) by its burrowing extension along the medullary portion of the bone. Usually, however, a dentigerous cyst involving the third molar is of the nature of a cyst of eruption, which has in its growth enveloped the unerupted tooth, or the tooth erupts into the cyst; such cysts may occur late in life and have been found up to sixty and sixty-five years (fig. 703). Should they contain deciduous teeth or permanent first molars, which is extremely rare, they are almost certainly caused by a cyst of eruption enveloping these teeth as it grows and extends; and the same remark usually applies when they contain a tooth or teeth in excess of the normal number, but in this latter case they approximate or even become identical with the cysts often found in connection with the multiple formations of compound composite odontomes.

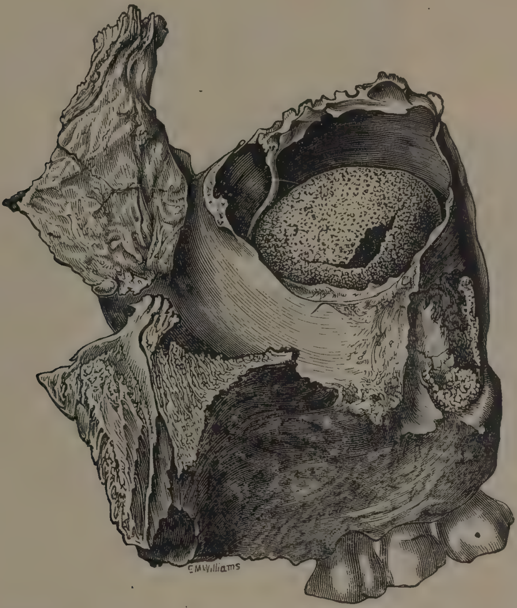


FIG. 702.—Calcareous matter in wall of dentigerous cyst. The cyst contained a supernumerary tooth. ('Dental Pathology and Surgery,' S. J. A. Salter.)

It must be remembered that a cyst may not enlarge fast enough to envelop an underlying unerupted tooth, and that the tooth may thereby become diverted and erupt, perhaps displaced; when this happens a simple epithelium-lined cyst is found in the jaws adjacent to the tooth.

Pathology.—The pathology of dentigerous cysts is identical with that of dental cysts, except that when in the course of their growth they come up against the crown of an unerupted tooth, being unable to cause its absorption or push it out of the way, they envelop it. Envelopment of the enamel-covered crown takes place readily as there is little resistance, but the forming periodontal membrane is attached to the tooth at its neck and this attachment offers some resistance to the enveloping process, so that often the crowns of teeth only are found projecting into the cavities of cysts (see fig. 701); in the absence of treatment this resistance is overcome and ultimately the whole tooth may be enveloped except that the apical portion usually remains attached.

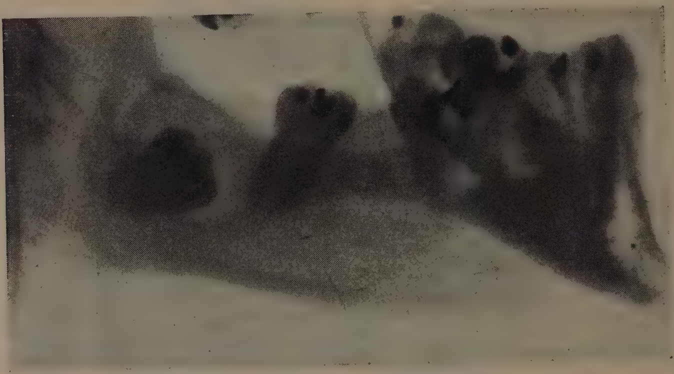
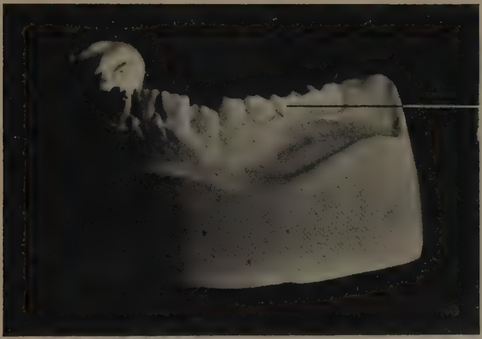


FIG. 703.—Cyst involving mandibular third molar.
(Stanley Colyer.)

Treatment.—Treatment should be along the same lines as that for dental cysts. If the age at which the cyst is treated is anywhere near that of the normal date for eruption of the tooth in the cyst, this tooth should be left *in situ*, even though its attachment appear slight; very shortly the adjacent tissues close in on it and support it, and as the obstruction to its eruption is now removed it will erupt and often take up its normal position in the arch without any further treatment (see figs. 704 to 710).

If the age of treatment is long past the date of normal eruption of the tooth involved or it has become grossly misplaced, it should be removed, but on occasion it may be helped to attain its normal position in the arch by means of an orthodontic appliance. See p. 714.



$\overline{|\mathrm{E}}$ carious, and with dead, infected pulp

FIG. 704.—E. C., male, aged $9\frac{1}{12}$ years, dentigerous cyst. (E. S.) (From *Proc. Roy. Soc. Med.*)
(Now in odontological collection at the Royal College of Surgeons of England.)

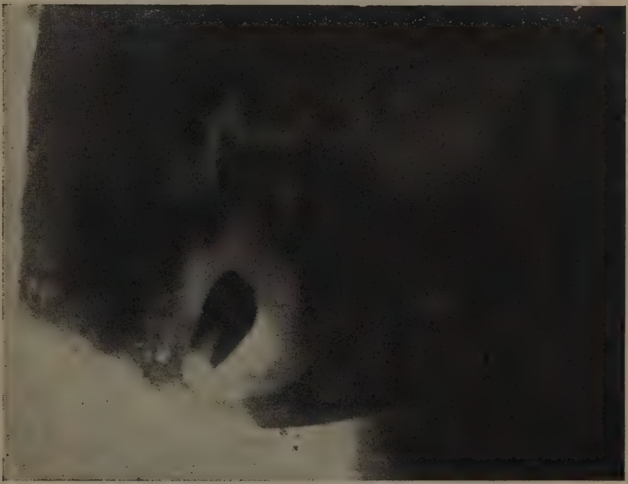


FIG. 705.—Radiogram of fig. 704. (E. S.) (From *Proc. Roy. Soc. Med.*)
(Now in odontological collection at the Royal College of Surgeons of England.)

NOTE.—From what has been said concerning these three types of unilocular epithelial cyst of the jaw, it appears that they are all due to proliferation of remnants of the epithelial dental formative organs, stimulated into activity by sepsis, delayed eruption or, possibly, trauma, and that their distinctive names depend chiefly

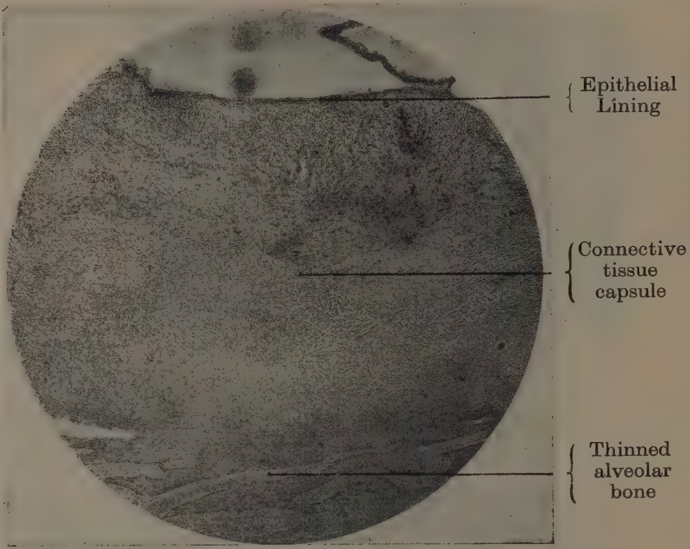


FIG. 706.—Photomicrograph of portion of cyst wall of fig. 704. (E. S.) (From *Proc. Roy. Soc. Med.*)

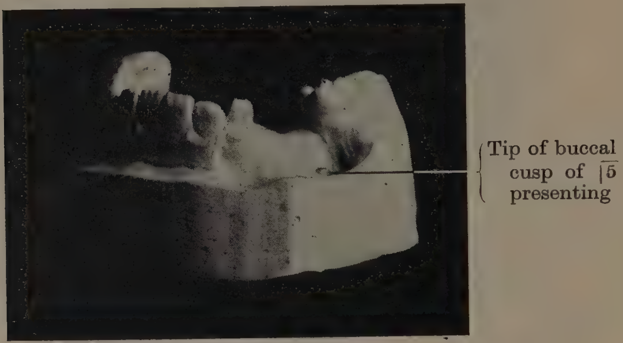
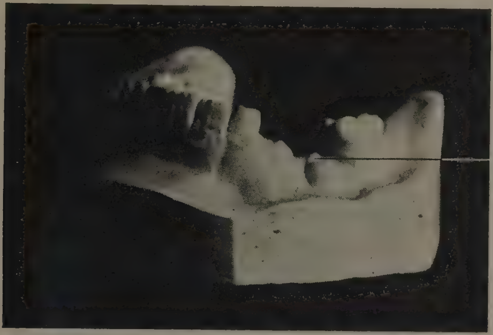


FIG. 707.—Fig. 704, twenty-nine days after operation. (E. S.) (From *Proc. Roy. Soc. Med.*) (Now in odontological collection at the Royal College of Surgeons of England.)

on (1) whether they are superficial to an erupting tooth (cyst of eruption); (2) whether they partly or wholly envelop an unerupted tooth (dentigerous cyst); (3) whether they are found in direct connection with a pulpless and infected tooth (dental cyst); (4) whether when seen they appear to be independent of teeth (epithelial-lined cyst of jaw).

Changes occasionally take place in the type of growth of the epithelial lining of dental and dentigerous cysts. (See p. 714.)



Tip of buccal cusp of $\overline{|5}$

Fig. 708.—Fig. 704, 133 days after operation. (E. S.) (From *Proc. Roy. Soc. Med.*)
(Now in odontological collection at the Royal College of Surgeons of England.)

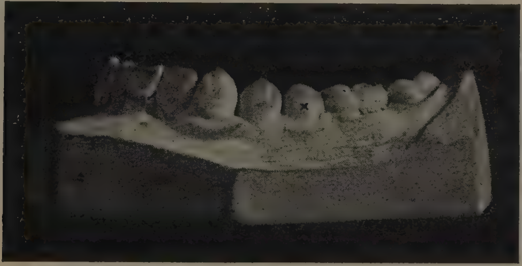


Fig. 709.—Fig. 704, four years after operation. $\overline{|5}$ in normal occlusion. (E. S.)
(Now in odontological collection at the Royal College of Surgeons of England.)

Other unilocular epithelial cysts occur in the maxilla which are not of dental origin. These are **naso-palatine cysts**[1] and are probably inclusion cysts and therefore dermoids, containing sebaceous material as with dermoids elsewhere; a similar type of cyst may also arise at the lines of adjacent developmental union of embryonic processes, and there is some evidence that occasionally they may be of the nature of retention cysts. Sometimes routine radiological examination gives the first intimation of their presence. They may become infected and then simulate alveolar abscesses and possibly they have often been mistaken for dental cysts in the past; glandular structures may be found in the epithelium lining them. If by reason of their size or infection they give rise to untoward signs or symptoms they should be removed; they usually shell out readily, but may be attached to the upper nasal portion of the naso-palatine canal above and the palatine mucosa below.

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[1] For a detailed account of Cysts of Developmental Origin in the Premaxillary Region, see H. T. Roper-Hall, *Brit. Dent. Journ.*, 1938, lxv, 405, and W. Brandt and H. T. Roper-Hall, *Brit. Dent. Journ.*, 1941, lxx, 213.

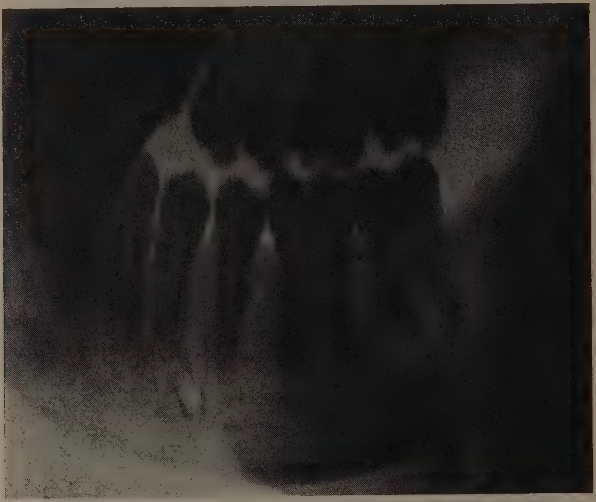

FIG. 710.—Radiogram of case shown in fig. 704, four years after treatment, showing completion of development of |5. (E. S.)
(Now in odontological collection at the Royal College of Surgeons of England.)

Occasionally, but rarely, unilocular epithelial lined cysts occur in the mandible, usually in the third molar region and quite apart from the presence of pulpless or unerupted teeth, and may contain a semi-solid yellowish-white material not unlike cerebral matter in appearance and feeling greasy to the touch. The origin and nature of these cysts is uncertain; glandular structures histologically identical with sebaceous or ceruminous glands have been described and figured [1] as occurring in the gum in the region of the third molar, and it is possible that these cysts may have

[1] *Dental Record*, 1933, 53, 369. (E. S. and W. R. Keizer.)

their origin from these structures. The frequency with which these glandular structures occur is unknown. It has also been suggested that they may be unilocular forms of the multilocular cyst, and in support of this view is the fact of their occasional early extension to the tip of the coronoid process.

Treatment.—They should be removed, and in view of the possibility of their recurrence in a multilocular form, which the writer (E. S.) has known to occur on several occasions, particular care should be taken to ensure the complete removal of the whole of the epithelial lining.

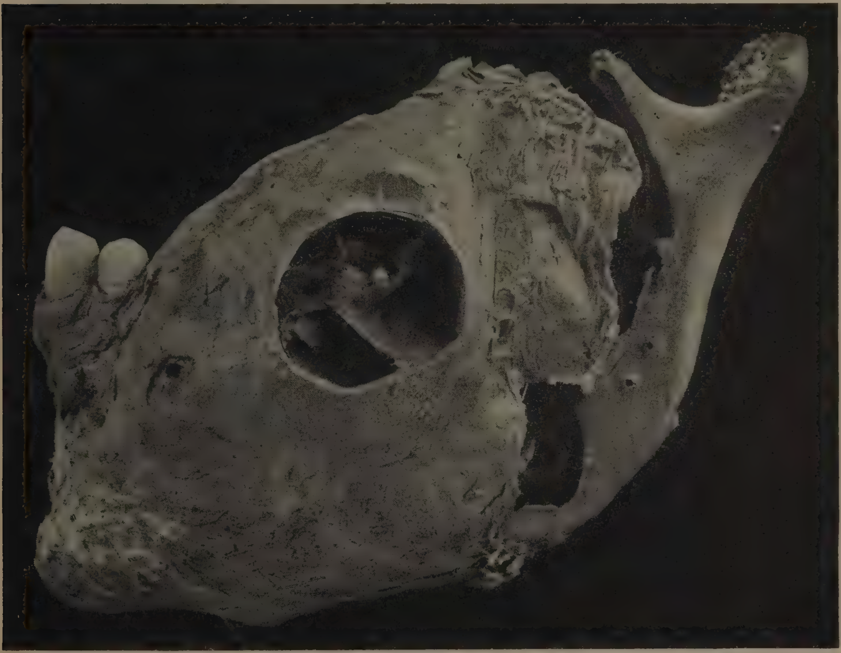

FIG. 711.—Left half of the mandible affected by a multilocular cyst, No. 2198, Museum Royal College of Surgeons of England.
(From the Report on Odontomes (*Brit. Dent. Assoc.*).)

(4) **Multilocular Cysts, Basal-celled Carcinomata, 'Adamantinomata,' or Epithelial Odontomes.**—These are found most frequently in the mandibular molar region, though they have occasionally been found in the maxilla and in the more anterior portion of the mandible. They are said to occur rather more frequently in females, but the sex difference as noted in a large number of cases in the United States is but small. They are usually first

noticed about the age of thirty, but occasionally in young children and also in old people.

Occasionally the whole of the normal molar series of teeth is present in the neighbourhood of the cyst, their development and occlusion being normal. Sometimes apparently the cyst has developed before the eruption of the third molar and prevented its eruption, or the tooth may be found in the substance of the cyst.

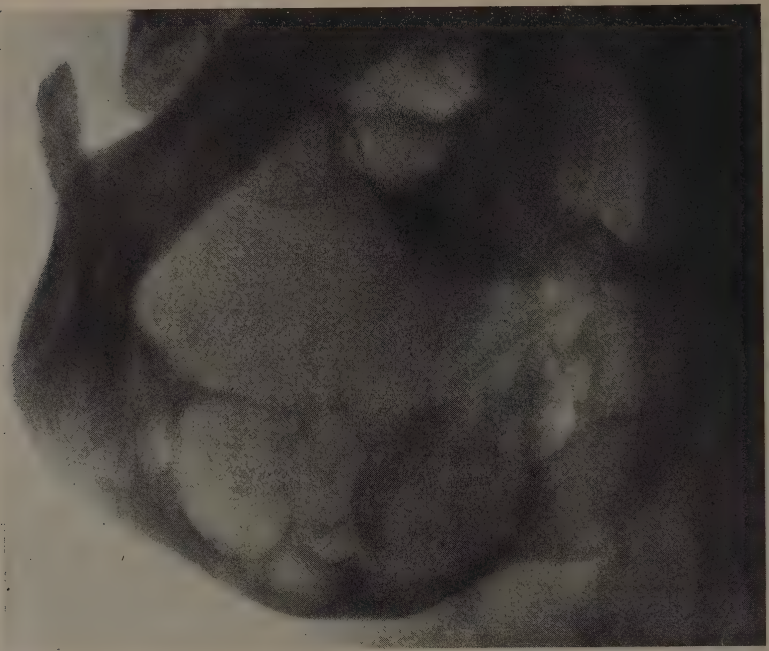

Fig. 712.—Radiogram of multilocular cyst of mandible. (W. R. Keizer.)

The swelling increases slowly and painlessly (fig. 711), shows up more prominently on the outer aspect of the mandible and presents in the mouth as a lobulated swelling, some of the lobules being occasionally fluctuant. Radiograms show several or many cysts more or less separated by partial or complete bony septa (fig. 712); they often extend into the coronoid process to its tip. As they increase in size they may extend downwards into the soft tissues of the neck and give rise to a very extensive mass. Some of them are solid rather than cystic, particularly when they have extended into soft tissues. At no time have calcified dental tissues been found in these cysts, though 'epithelial pearls' or cell nests, sometimes termed 'enameloid,' have been described in two or three cases. Their growth has been known to extend for over thirty-five years. They are neoplasms, and do not appear to come within the same category as the unilocular cysts at all, but to be of the nature of basal-celled carcinomata, usually of the gum. Although these growths are usually single, McGregor[1] records ten cases in which metastases had occurred; in one of these recorded by Vorzimer and Perla[2] the metastases were in the lung.

Histologically, they are found to be made up of a number of cysts of varying sizes, but seldom exceeding 2 cm. in diameter.

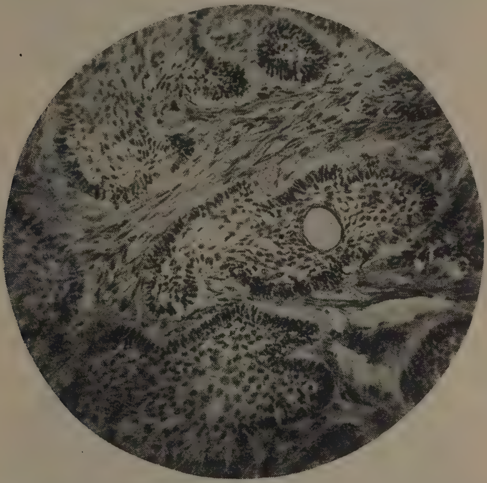

Fig. 713.—Section of multilocular cyst of mandible showing cystic area and areas of cells resembling stellate reticulum. × 100. (W. R. Keizer.)

The cysts are separated by fibrous septa which are sometimes ossified. Structurally, they consist of rounded cavities or columns of epithelium forming alveoli, bordered in each case by a basal layer of columnar or spheroidal epithelium and occupied either by a glairy yellowish or brownish fluid or by epithelium in a state of degeneration, more or less advanced, and which often appears like the stellate reticulum of the enamel organ (figs. 713 and 714). The mass is surrounded by a fairly firm capsule.

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[1] *Acta Radiologica*, Stockholm, 1935, 16, Fasc. 2, 254.

[2] *Dental Cosmos*, 1932, lxxiv, 1232.

Origin.—The coincidence of cells exactly resembling the stellate reticulum lying adjacent to a columnar epithelium has caused the latter to be likened to the ameloblast cells of the enamel organ, and the conclusion to be drawn that these cysts

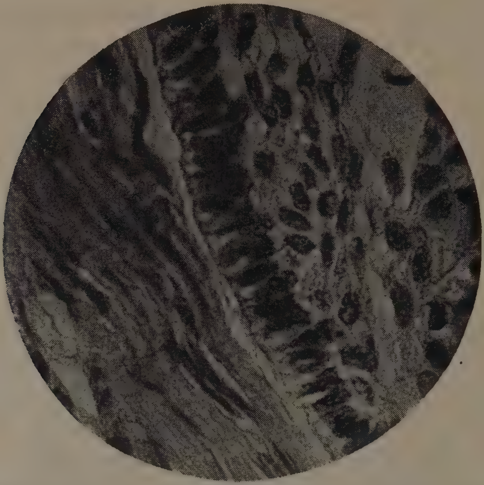

Fig. 714.—Section showing columnar cells of multilocular cyst of mandible. × 470. (W. R. Keizer.)

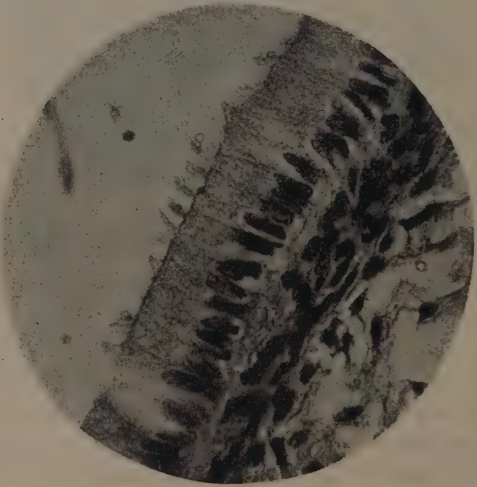

Fig. 715.—Section showing human ameloblasts. × 470. (W. R. Keizer.)

are therefore aberrations of the enamel organ, but the columnar cells are not like those of the enamel organ, either in size (figs. 714 and 715), shape, the position of the nucleus, or the presence of Tomes's process ; moreover, other components of the enamel organ are missing, though sometimes obliquely cut columnar cells may somewhat resemble the stratum intermedium ; sometimes all the normal teeth are present, and in cases where they are not it is often impossible to get an accurate history as to their removal or congenital absence, so that what evidence one has on this point indicates rather that they do not have origin from enamel organ cells.

Owing to the facts that these cells sometimes bear some resemblance to those of the normal enamel organ and that these growths occur chiefly in the jaws, it has been concluded that they originate from the formative dental epithelium ; but it seems likely that only a small minority do so. According to McGregor [1] some show histological evidence of origin from the gingival epithelium, and all appear to be connected with it.

The age incidence as usually given, 30 to 40 years, is stated to be an over-estimate, although this is often the age when they are first noticed ; they are distinctly rare in the young, and this caused Bland-Sutton [2] to remark that 'if they arise in epithelial vestiges of the enamel organ, they ought, theoretically, to be met with in the young.' A few cases have been described as occurring in childhood ; there is, however, a margin for error in the histological appearances seen at that period. If, for example, a complex composite odontome is seen in an early developmental stage and before any calcification has commenced, there is every incentive to describe it as an adamantinomatous growth, an error which its further development could alone rectify.

There is no case on record of fully formed enamel alone being found in these tumours, and the name 'adamantinoma' necessarily implies that it should occasionally be so present. Under normal circumstances enamel is never laid down except on a preformed dentine base, and this is common to the whole vertebrate world. The question naturally arises : Can enamel be laid down other than on a dentine base ? If it is so in these tumours, then such occurrence is unique ; those in which it has been laid down with or on dentine should of course be grouped with the composite odontomes.

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[1] *Acta Radiologica*, Stockholm, 1935, 16, Fasc. 2, 254.

[2] 'Tumours Innocent and Malignant,' London, 1922, 7th ed., 237.

The formation of an enamel organ invariably precedes that of a tooth, whether any enamel is ultimately formed on it or not. Huggins, McCarroll, and Dahlberg [1] in 1934, by the method of transplanting developing dental tissues, found that enamel was deposited only on dentine, though dentine could be laid down independently of enamel, but only when odontoblasts were present ; more recently Glasstone [2] by growing portions of tooth germs *in vitro* finds that the ameloblast layer is essential for the forming of odontoblasts, and otherwise confirms the work of Huggins, McCarroll, and Dahlberg. It would seem, therefore, to be presumptive evidence that because odontoblasts do not become differentiated in these growths, the basal cells of these tumours are not ameloblasts even though they may occasionally somewhat resemble them. Elsewhere [3] attention has been drawn to the histological and clinical similarities between this tumour and the basal-celled carcinomata of skin (rodent ulcers) and some of the epithelial tumours of the anterior (suprasellar) portion of the pituitary body. Duffy,[4] and Critchley and Ironside [5] describe these last tumours as pituitary adamantinomata, a term first applied to them by Onanoff in 1892.[6] A comparison of sections of these three types of growth at once emphasises their similarities, and if a portion of a multilocular cyst from the jaws be reported on in a pathological laboratory it will usually be returned as a basal-celled carcinoma.

One of us (E. S.) has seen a case involving soft tissues only on the lingual aspect of the mandible in the third molar region, the bone being entirely unaffected.

Several cases are on record where, after trauma has been applied to the skin, as for instance over the tibia, tumours histologically identical with multilocular cysts have developed and been described as adamantinomata. It is farcical to call them by this name, as dental formative epithelium has never been known to occur in such a site.

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[1] *Journ. Exper. Med.*, New York, 1934, 60, 199.

[2] *Journ. of Anat.*, London, 1936, 70, Pt. 2, 260.

[3] *Dental Record*, London, 1933, 53, 369. (E. S. and W. R. Keizer.)

[4] *Annals of Surgery*, 1920, 72, 537–555 and 725.

[5] *Brain*, London, 1926, 49, Pt. 4, 437.

[6] 'Sur un cas d'epithelioma,' *Thèse de Paris*, 1892 (cited by W. C. Duffy, *Annals of Surgery*, 1902, 72, 745).

When multilocular cysts are present in the jaws, quite often the whole of the normal series of teeth is present ; so it does not look as if any tooth germ of the normal series is responsible for them, and though it has been thought that they may originate from the germs of supernumerary teeth, it must be remembered that these occur far more commonly in other parts of the jaws where multilocular cysts are but rarely seen.

In view of its histological and clinical affinities it would seem more logical to call the usual types of this growth basal-celled carcinomata of the jaw ; and the tumours used for comparison : basal-celled carcinomata of the pituitary body, and skin overlying the tibia respectively, just as the rodent ulcer is now termed a basal-celled carcinoma of skin.

These same basal cells, however, also give rise to the dental formative epithelium, and a similar aberration of growth may also occur there, and presumably at any stage of its development. That being so, it seems probable that the ultimate product will depend on the stage of dental development at which the aberrant growth commences ; and that when it commences after the differentiation of odontoblasts, composite odontomes will be produced ; and in earlier stages and in the lack of odontoblast formation, growths of the usual type, or possibly containing granules representing a precursor of enamel only (*i.e.* potential adamantinomata). This, too, would account for the variable histological characteristics of some of these tumours.

It is conceivable, however, that when the aberration originates in the basal epithelium of the gum, it may occasionally attempt to consummate the ancestral tooth-forming function of this layer, and give rise to tumours comparable to those arising from dental formative epithelium. There is also the very recent evidence [1] that in one case the aberration apparently originated in the wall of a dental cyst, itself presumably originating from the rests of Malassez.

Etiology unknown, but similar growths have on several occasions been known to occur at the site of preceding trauma, in the tibia for example ; and there has occasionally been a history of trauma when they have occurred in the jaws.

Pathology.—In their growth they cause an osteoclastic erosion of the bony mandible extending primarily along the cancellous portion and particularly to the tip of the coronoid process; the eroded bone is replaced by the growth; they invade soft tissues also, and as a rule give rise to no metastases.[1] Occasionally they ulcerate in the mouth. The cyst contents are apparently derived from the central epithelial cells which have undergone hydropic degeneration in the same manner as do the central cells of dental cysts. It is probable that the ossification of the fibrous septa and the occurrence of bony trabeculæ in such portions of these tumours as occur in the jaw are merely due to their environment and the fact that the growth and stability of bone are interfered with, and that it sends out these trabeculæ in an effort of repair or reaction, as they do not occur in those parts of the tumour not having a bony environment.

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), 1938, xxxi, 1138. (G. T. Hankey).

Treatment.—If they are small, treatment may consist in the removal of the cyst via the mouth, care being taken that all the loculi are opened and their contents and lining removed. Such treatment, however, is not very often successful, as recurrences are frequent, and in some cases recurrence has taken place after ten or more operations.

More satisfactory results have been seen when the portion of jaw containing the cyst has been excised and the two portions of jaw retained in their correct position by means of a metal cap splint, made before operation, till such time as healing has taken place, when a bone graft from the iliac crest is inserted to replace the portion of jaw removed. When such a splint is impracticable, as often occurs, since the growth commonly involves the molar region extensively, metal cap splints are made to cover both the maxillary and the mandibular teeth, and the larger portion of the mandible is splinted into correct occlusion with the maxilla till healing has taken place and the bone graft can be put in. It is probable that many cases described in the past as being spheroidal or columnar carcinomata were really tumours of this nature. In cases of doubt a small portion should be removed for biopsy. (See "Biopsy," p. 989.)

Occasionally, but rarely, **changes may take place in the type of growth of the cells lining both dental and dentigerous cysts.**

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[1] Vorzimer and Perla report an instance of 'adamantinoma' of the jaw with metastases to the lung. The bronchi of the lower lobe were markedly dilated and their lumina filled with a cast of the tumour tissue. In places the parenchyma of the lung was invaded. The authors suggest that the tumour tissue was aspirated into the lung from the primary tumour by way of the trachæa and bronchial tree, and grew primarily within the lumina of the bronchi (*Dental Cosmos*, 1932, lxxiv, 1232).

There are at least eight cases on record of what has clinically and radiologically appeared to be the usual type of dental or dentigerous cyst, but microscopical examination has shown that at one part of the cyst wall a change has taken place in the character of growth of the epithelium, which seems to have taken on a neoplastic character such as is seen in multilocular cysts.

So far as is known this change, though it may be obvious to the naked eye after the removal of the cyst lining, has not yet been diagnosed before treatment. These cases emphasise the importance of the pathological examination of tissues after removal, particularly when the cyst lining shows local differences in appearance. One such case is recorded by G. T. Hankey in *Proc. Roy. Soc. Med.*, 1938, xxxi, 1138, and another is described and figured (fig. 14) in the 'Report on Odontomes,' 1914 ; others occur in the American literature and the writer has had one which made itself known as a recurrence in multilocular form. When this change occurs there is a neoplasm originating from the dental formative organs, *i.e.* an (epithelial) odontome.

CHAPTER XXII

Odontomata and Abnormal Productions.

Composite and Connective Tissue Productions.

(B) COMPOSITE PRODUCTIONS.

(1) ARISING FROM TOOTH GERMS OF THE NORMAL SERIES.

(a) Adichotomous Abnormalities.—These are teeth with aberrant crowns or roots outside the range of normal variation, and hence imperfectly co-ordinated. They are nearly all of subnormal growth, and are exemplified by small teeth or denticles in place of and representing teeth of the normal series (figs. 716 and 717). Usually both the crown and root portions are affected and only one tooth, usually in the permanent dentition.

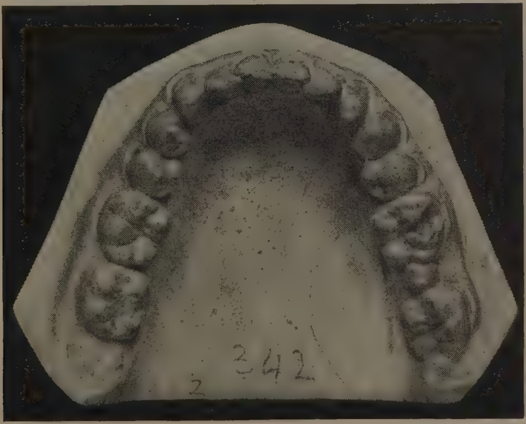


FIG. 716.—Model showing denticle representing $\overline{7}|$ and nearly fully erupted $\overline{8}|$. $|\overline{8}$ is erupting. (E. S.)

Treatment.—If unsightly, functionless, or in any way interfering with occlusion, they should be removed.

Some partly unco-ordinated forms must also be admitted under this heading, as in some dilated odontomes there appears to be no attempt at dichotomy (division) of the tooth germ, but merely an invagination caused primarily by unco-ordinated epithelial growth. This matter is further discussed under the pathology of dilated odontomes on p. 733 *et seq.*

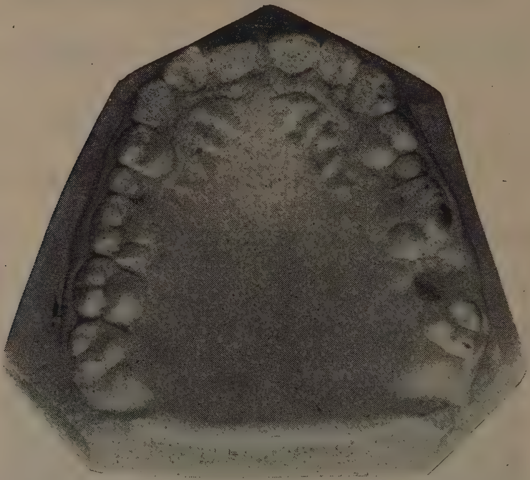


Fig. 717.—Model showing symmetrical denticles representing second premolars, female. (E. S.)

(b) Incomplete Dichotomies.—There are many varieties of these and they can readily be grouped according to the degree of co-ordination in their growth and the extent to which the tooth germ is involved, as follows :—

(i) **Co-ordinated : of part of the tooth germ.**—This results in the formation of extra cusps or roots ; they are examples of co-ordinated incomplete dichotomy, as they represent attempts at division of that part of the tooth germ forming the crown or root respectively. It might be thought that roots should be grouped under the connective tissue abnormalities, but as epithelium is intimately concerned in the formation of roots, the sheath of Hertwig outlining and governing their growth and extent (fig. 718), they must obviously be grouped under the composite abnormalities. Actually, the very great majority of abnormalities of this nature come within the range of normal variation.

Treatment.—No treatment is necessary.

Of the whole tooth germ.—These occur, either as diminished growth, in that two (or more) tooth germs of the normal series are developmentally united, so giving the appearance of incomplete equal dichotomy (gemination), or as increased growth, in that a tooth germ of the normal series attempts to divide and form two similar teeth—this is an actual *incomplete equal dichotomy* (see figs. 731 to 734). Sometimes these conditions of increased and diminished growth are coincident, as in figs. 723 and 724; this condition is rarer in the permanent dentition (see fig. 751).

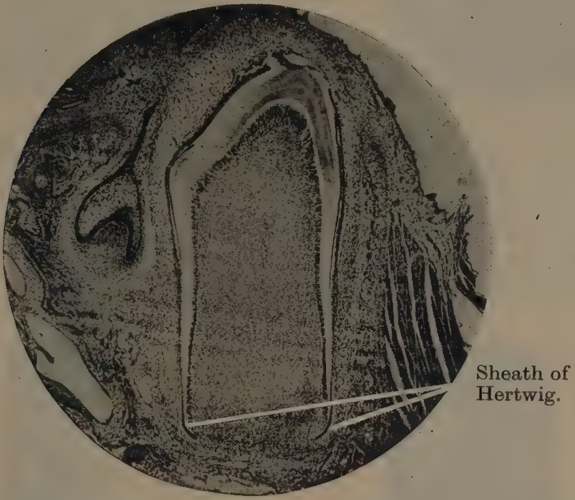


FIG. 718.—Section showing epithelial sheath of Hertwig, a continuous sheath at this stage. (E. S.) (From *Brit. Dent. Journ.*)

Should it be considered undesirable to consider any of these productions as decreased growth, one may, taking figs. 725 and 726 as examples, regard the second incisors as being congenitally absent and the productions figured as being incomplete equal dichotomies of the first incisors.

Incomplete equal dichotomy varies considerably in the intimacy with which the teeth are joined; sometimes the component teeth may be clearly distinguished, but sometimes union is so complete as to mask the identity of the components. From this it may be gathered that the pulp chambers may be separate or conjoined throughout their extent. Union may be restricted to the crowns or the crowns may be separated and the roots united.

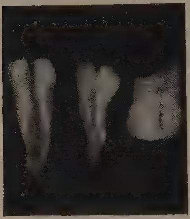


FIG. 719. FIG. 720. FIG. 721.

FIG. 719.—Union of mandibular deciduous second incisor and canine, decreased growth.

FIG. 720.—Union of mandibular deciduous first and second incisors, decreased growth.

FIG. 721.—Union of maxillary deciduous first and second incisors, decreased growth.

(Sir Francis Farmer.)

(From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

It occurs both in the deciduous and permanent dentitions :—

(*a*) *In the deciduous dentition* it occurs in the incisor region of both maxilla and mandible—though more commonly in the latter ; in the mandible the first and second incisors, or more commonly the second incisor and canine, may be joined (figs. 719 to 722). It has not yet been recorded in the molar region, but has been seen in the maxillary canine. Joined deciduous teeth may or may not be succeeded by a similar condition in the permanent teeth or a tooth may be absent from the permanent series—all such variations have been recorded. It occurs rather more commonly in this than in the permanent dentition and is usually asymmetrical.

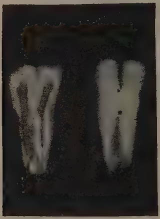


FIG. 722.—Union of mandibular deciduous second incisor and canine and the same in section to show the pulpal distribution, decreased growth.

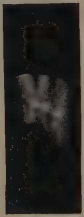


FIG. 723.—Union of mandibular deciduous first and second incisors with an incomplete dichotomy joining both and resembling a mandibular incisor.

(From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

Occasionally, two teeth of the normal series are associated with an incomplete dichotomy which joins all, so that an appearance of three teeth joined is produced (fig. 723), and in one case this has been known to be symmetrical (see fig. 724).

In the permanent dentition, it is commonest in the maxillary and mandibular incisor and molar regions, but owing to the different ages at which development takes place is very rare between premolars and molars. It does not occur in the canine regions as a form of diminished growth, possibly because these teeth are developed at some distance from those which will ultimately lie adjacent to them, but it does in a form of increased growth (see figs. 731 and 734). It is frequently, but not always, symmetrical, especially in the maxillary incisor region. It is rare in the premolar region (figs. 725 to 734).

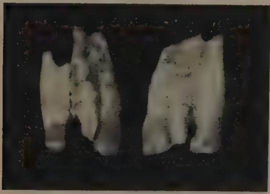
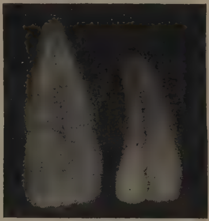


Fig. 724. — Symmetrical union of maxillary deciduous first and second incisors with an incomplete dichotomy joining both. (London Hospital Dental Museum.) (E. S.)



Figs. 725 and 726.—Different degrees of union of maxillary permanent first and second incisors, decreased growth. (Sir Francis Farmer.) (From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

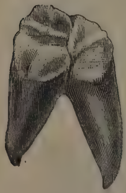


Fig. 727.—Union in maxillary incisor region involving coronal portions of teeth only, decreased growth.

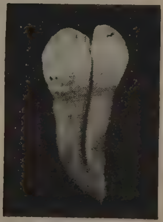


Fig. 728.—Union of two maxillary premolars, decreased growth.

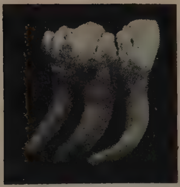


Fig. 729.—Union of mandibular permanent first and second molars, decreased growth.

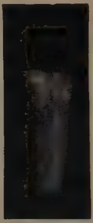


Fig. 730.—Union of two mandibular permanent incisors of the normal series, decreased growth. (From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

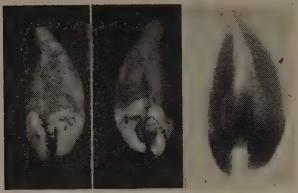


Fig. 731.—Incomplete equal dichotomy of maxillary left permanent canine: labial, palatal and radiographic views. The pulp canals become confluent; increased growth. (E. S.)

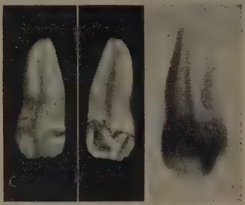


Fig. 732.—Incomplete equal dichotomy of maxillary left permanent second incisor; labial, palatal and radiographic views. The pulp canals are separate and the tendency to bifidism is more obvious palatally than labially. Increased growth. Female. (E. S.)

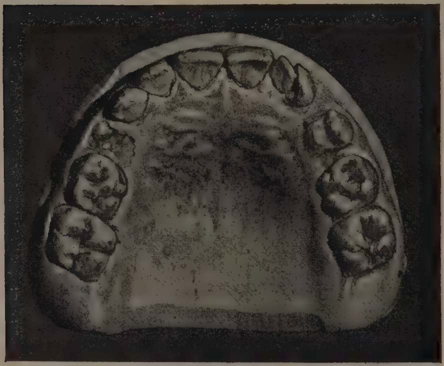


Fig. 733.—Incomplete equal dichotomy like a tooth of the normal series, joined, in the root portion only, with the second incisor. Increased growth. An unerupted cone-shaped tooth was also present. The mother and two aunts each had an extra incisor in the left maxilla.

Treatment.—Quite often incomplete equal dichotomies are in good occlusion and function well, and so should be left *in situ* ; in the front of the mouth they may be unsightly, and the merits of each case must then guide one as to whether they should be removed or not.

(ii) Imperfectly Co-ordinated.—Incomplete dichotomies of this type occur as forms of increased growth of different degrees according to the site and extent of the growth produced ; they may appear as enamel nodules, which are due to aberrant growth of part of the tooth germ only and are really aberrant cusps ; or extra cusps and roots may be in association with each other so as to look like an additional tooth joined to one of the normal series ; actually this is an *incomplete unequal dichotomy*. In the great majority of this latter type the greater part of the production resembles the normal, in that it looks like a normal tooth with a denticle joined on to it, but occasionally neither or none does so (see figs. 752 to 756).

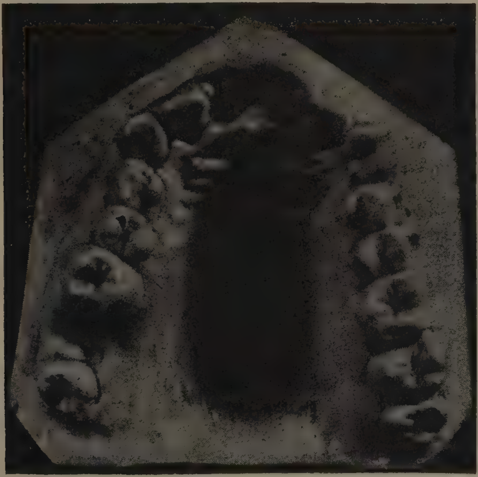


FIG. 734.—Maxillary left permanent canine with bifid crown, female, increased growth. (E. S.) (From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

According to the degree and position of the abnormality they may be grouped as follows :—

Of part of the tooth germ. Enamel Nodules.—These consist of a core of dentine covered by a thick cap of enamel (fig. 735) and vary in size from a minute speck to that of a large cusp ;

when large they contain a cornu of pulp. They occur chiefly at the junction of the roots of multi-rooted teeth and are sometimes in continuity with the coronal enamel by a thread-like lamina of enamel, though often they are isolated; more rarely they are in gross continuity with the coronal enamel (figs. 736 to 745).

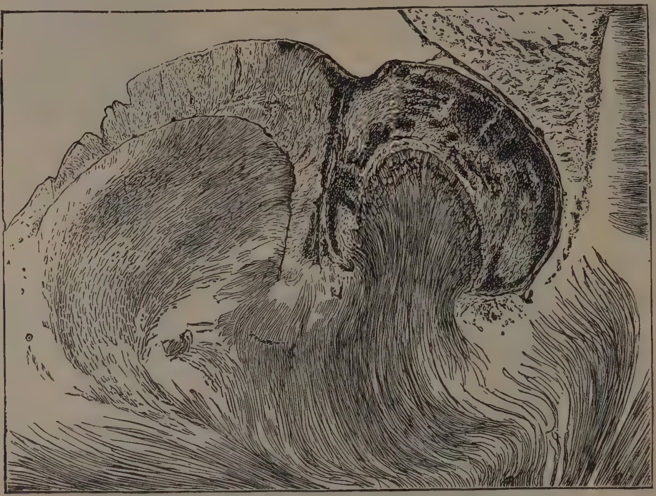


FIG. 735.—Section through an enamel nodule upon a maxillary second molar.

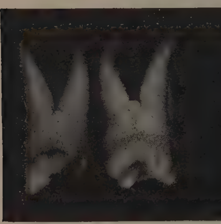


FIG. 736.

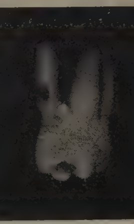


FIG. 737.

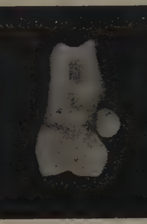


FIG. 738.

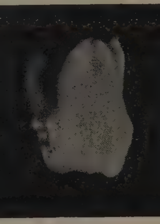


FIG. 739.

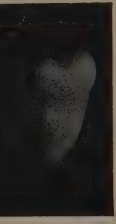


FIG. 740.

FIG. 736.—Symmetrical maxillary permanent first molars from same mouth with symmetrical enamel nodules in the common site between palatine and posterior buccal roots. (One of these teeth has a second enamel nodule between the two buccal roots.) Female.

FIG. 737.—Maxillary permanent first molar with enamel nodule in the common site between palatine and posterior buccal roots.

FIG. 738.—Maxillary molar with large enamel nodule.

FIG. 739.—Maxillary molar with large enamel nodule confluent with coronal enamel.

FIG. 740.—Mandibular molar with large enamel nodule.

(736 and 737, E. S.; 738, 739 and 740, Sir Francis Farmer.)

(From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

They also occur on single-rooted teeth; sometimes there is more than one on the same tooth and occasionally they are symmetrical (fig. 736). They are very rare on deciduous teeth (fig. 745). The great majority of enamel nodules occur at the junction of the palatine and posterior buccal roots of maxillary permanent molars.

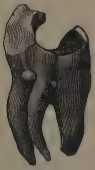


FIG. 741.—Maxillary molar with two enamel nodules.

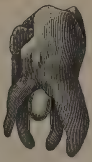


FIG. 742.—Maxillary molar showing large enamel nodule.

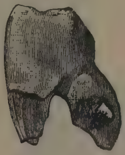


FIG. 743.—Maxillary molar with enamel nodule near apex of root.

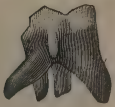


FIG. 744. — Prolongation of enamel between the roots of a maxillary molar.

Origin. — The facts that many enamel nodules have a direct enamel connection with the coronal enamel, that they are very constant in their position and that they may be symmetrical, indicate that they vary only in degree and position from extra cusps and therefore have origin from the enamel organ, and in some cases it is difficult to decide whether a nodule should be classed as an extra cusp or not. S. J. A. Salter, in 1874, described them as submerged cusps (see p. 754).

FIG. 745.—Symmetrical maxillary deciduous second molars showing large and symmetrical enamel nodules on buccal aspect, and extra buccal cusps on one of them. (E. S.) (From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

Treatment.—As they give rise to no signs or symptoms no treatment is necessary; indeed, they are rarely diagnosed till the tooth they are attached to is extracted, and only on close inspection then. When they are connected with the coronal enamel by a thin enamel lamina there is necessarily a potential 'pocket' leading to the nodule, which may be of clinical importance.

Of the whole tooth germ.—This is shown by the association of additional cusps and roots with each other giving the appearance of an extra denticle unlike any of the normal series joined on to a normal tooth, and is actually an *incomplete unequal dichotomy.* In different specimens the gradation from extra cusps or enamel nodules, with no change in the roots of the teeth, to their support by an additional root outgrowth may be well seen. Though these forms occur in both dentitions they are rare in the deciduous dentition.

In the deciduous dentition it may occur in the maxillary and mandibular incisor regions.

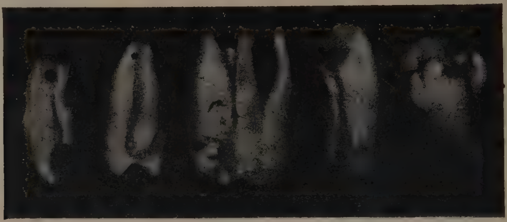


FIG. 746. FIG. 747. FIG. 748. FIG. 749. FIG. 750.

FIG. 746.—Maxillary permanent second incisor with an outgrowth like a conical tooth.

FIG. 747.—Maxillary premolar with an outgrowth like a conical tooth.

FIG. 748.—Maxillary permanent molar with a molariform outgrowth.

FIG. 749.—Mandibular permanent canine with an outgrowth like a slender denticle.

FIG. 750.—Mandibular permanent molar with a molariform outgrowth.

(746 and 750, E. S.; 747, 748 and 749, Sir Francis Farmer.)

(From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

In the permanent dentition it is commonest in the maxillary incisor region and the maxillary and mandibular molar regions and occurs occasionally in the premolar region. It occurs in both maxillary and mandibular canine regions, and when one considers the distance from other teeth at which these teeth are developed this indicates that it is definitely an incomplete unequal dichotomy of the canine tooth germ (fig. 749). It varies considerably in degree and in many cases it is difficult to decide whether a particular specimen should be classed as of this type of dichotomy or as a tooth presenting an extra cusp, the difference being merely one of degree and the dividing line arbitrary and relatively unimportant (see figs. 746 to 750).

Pathological Union of Teeth.—As the union is here solely by the connective tissue cementum this variety is discussed under the heading of Connective Tissue Odontomes—Cementomata.

Rarely the confusion and exuberance of dichotomous growth involve more than one tooth, as in fig. 751, where two teeth of the normal series are formed in one mass with productions resembling two other teeth; co-ordination, though imperfect, is not absent, and there is no neoplastic growth.

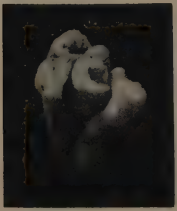


Fig. 751.—Two maxillary premolars joined with an outgrowth resembling two other teeth.

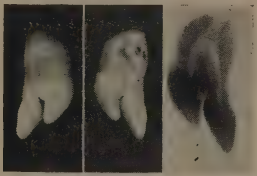
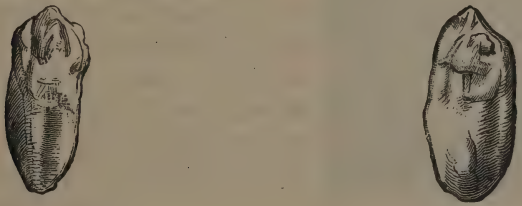


Fig. 752.—Imperfectly co-ordinated incomplete dichotomy in place of maxillary right permanent first incisor; palatal, labial and radiographic views. Neither component is like the normal; there are separate pulp canals confluent at the apex. (E. S.)

Occasionally neither or none of the components of the formation is like a tooth of the normal series (fig. 752), and more rarely still a tooth or more of the normal series may be represented by an imperfectly co-ordinated mass or formation which, on section, may be found to resemble several mis-shapen teeth joined together (figs. 753 to 756). Sometimes it is difficult to differentiate exactly between some of these formations and such small degree of unco-ordinated growth as occurs in some dilated odontomes without sectioning the specimen.



Frontal view. Lateral view.

Fig. 753.—Apparent union of three mis-shapen denticles replacing maxillary first incisor; it had three distinct pulp cavities.

An appearance of nine joined denticles has been found in one specimen; sometimes each denticle contains a separate pulp chamber, but sometimes a common pulp chamber is found. This type occurs most often in the maxillary incisor region and in the permanent dentition only, though they have also been found in the premolar and molar regions. They are comparable to complex odontomes and the difference between the two is probably only one of degree.

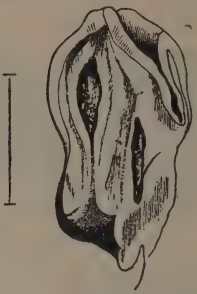


Fig. 754.—Section of fig. 753.

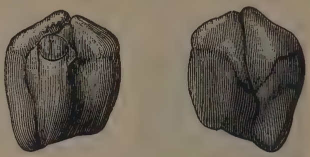


Fig. 755. Fig. 756.

Imperfectly co-ordinated incomplete dichotomy from maxillary first incisor region, apparently composed of three conjoined teeth; the second incisor was present. (From *Brit. Dent. Journ.*)

Very rarely the dichotomous portion may show some unco-ordinated growth, as in fig. 756a.

Treatment.—Incomplete unequal dichotomies are often in good occlusion and function well, and so may be left *in situ*; in the front of the mouth they may be unsightly, and the merits of each case must then guide one as to whether they should be removed or not. When neither or none of the components is like a tooth of the normal series they should usually be removed, as these are often both functionless and, if in the front of the mouth, unsightly.

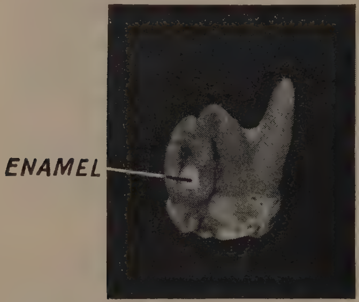


Fig. 756a.—Incomplete unequal dichotomy of maxillary left permanent second molar; the dichotomous portion is of partly unco-ordinated growth, *i.e.* a dilated odontome. (M. Porter.)

(iii) Partly Unco-ordinated. Most Dilated and some Complex Odontomes.—These consist mainly of what are called dilated odontomes, but some complex odontomes must be included here as a few of them show some, though imperfect, co-ordination as well as unco-ordinated growth, as may be seen in fig. 778. This matter is further discussed under Complex Odontomes on p. 735. In dilated odontomes one portion of the formation, either crown or root portion, but more often the latter, shows a disproportionate growth which may be very marked. They occur mainly towards the front of the mouth and in the maxilla, but also in the molar region, especially in the mandible.

When they occur in the crown they always show that during their formation there has been an irregular invagination of the enamel organ; this fact was brought out by one of us (J. F. C.) in 1926[1] and recently enlarged by Martin Rushton.[2] Occasionally there may be two invaginations of the enamel organ, and sometimes it appears as if one tooth had been superimposed or invaginated into another—these latter have been termed gestant odontomes. Dilated odontomes are frequently hollowed out on their deep aspect and present there a nutrient opening often considerably larger than the normal apical foramen; the hollowed-out portion is sometimes occupied by bone. The coronal portion may be almost normal in appearance and resemble a tooth like those in the neighbourhood in which it is found, or be cone-like on section; the coronal portion may be made up of normally arranged calcified dental tissues, the dilated portion being composed of normal dental tissues very much flattened out and in parts attenuated; sometimes, however, the arrangement is not normal, and enamel may be found on the root portion, in the hollowed-out portion, or even within the substance of the other calcified dental tissues (figs. 757 to 776).

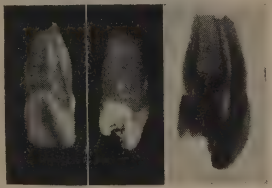


FIG. 757.—Dilated odontome in place of 1|. Labial, lateral and radiographic views. Labially the crown is like that of 1|. There appears to be an imperfectly co-ordinated incomplete dichotomy, and between it and the tooth there is an unco-ordinated enamel invagination. (E. S.)

Pathology.—The reason for this invagination of the enamel organ is not clear; Rushton, on the ground that the enamel organ is to all intents a secreting gland, has suggested tentatively that it may be likened to an adenomatous (*i.e.* neoplastic) production; presumably its ultimate growth being limited by its

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), 1926, 19, 39.

[2] *Guy's Hosp. Gaz.*, 1936, 50, 438.

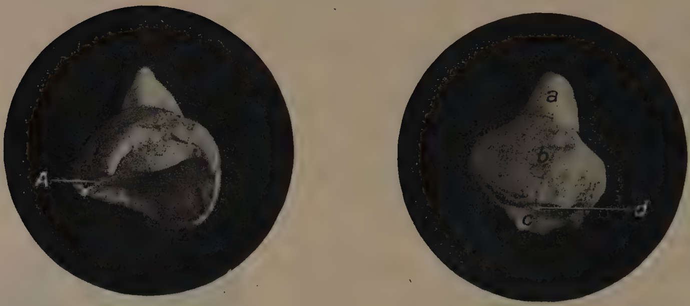
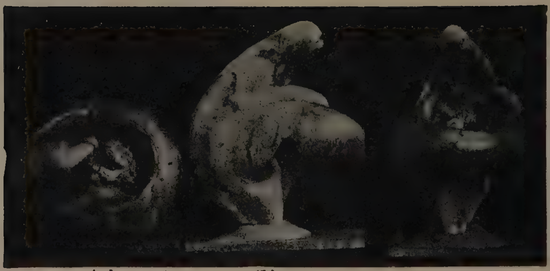


FIG. 758.—Dilated odontome.
(Graves Morris.) (From *Brit. Dent. Journ.*)



(*a*) (*b*) (*c*)

FIG. 760.—Dilated odontome: *a*, view of the upper aspect; *b*, view of the lateral aspect; *c*, view of the posterior aspect. The coronal portion is like that of a maxillary first incisor. (From *Trans. Odonto. Soc.*)

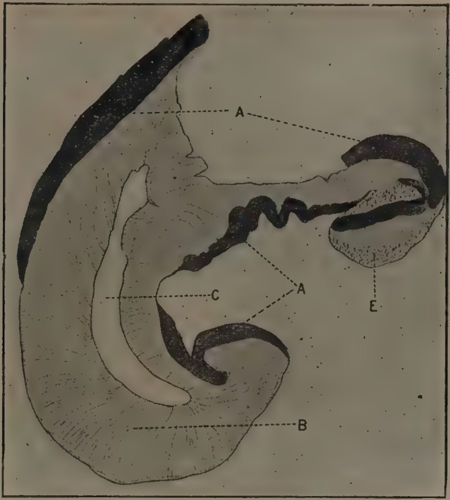


FIG. 761.—A, enamel; B, dentine; C, pulp cavity; E, cement. (Section of fig. 760.) (From *Trans. Odonto. Soc.*)

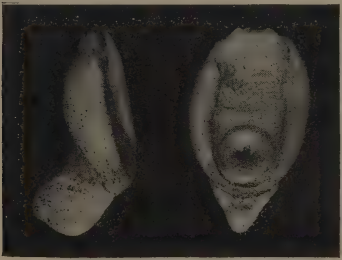


FIG. 762.—Dilated odontome from maxillary second incisor region.

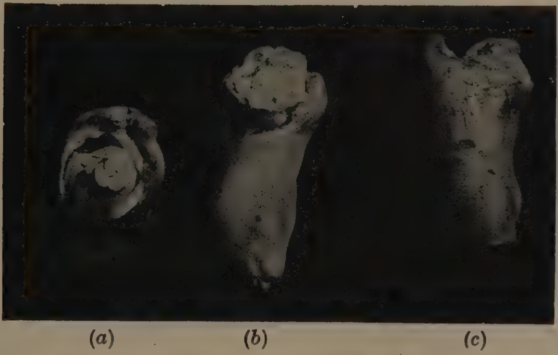

(*a*) (*b*) (*c*)

FIG. 763.—Dilated odontome from a male aged 17: *a*, view from base; *b*, view from posterior aspect; *c*, lateral view. From *Trans. Odonto. Soc.*)

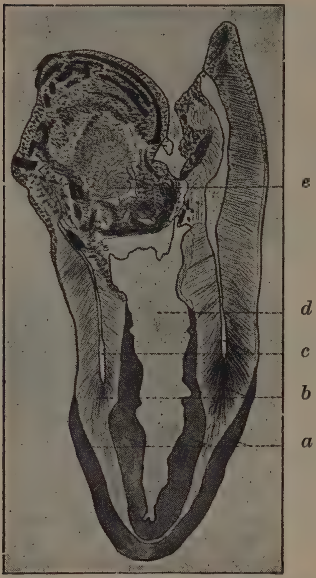


FIG. 764.—*a*, enamel; *b*, dentine; *c*, pulp cavity of external tooth; *d*, cavity lined with enamel; *e*, mass of vascular cement-like tissue. (Section of fig. 763.) (From *Trans. Odonto. Soc.*)

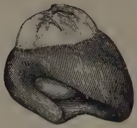


FIG. 765.—Dilated odontome removed from the region of the mandibular second molar. (From *Brit. Dent. Journ.*)

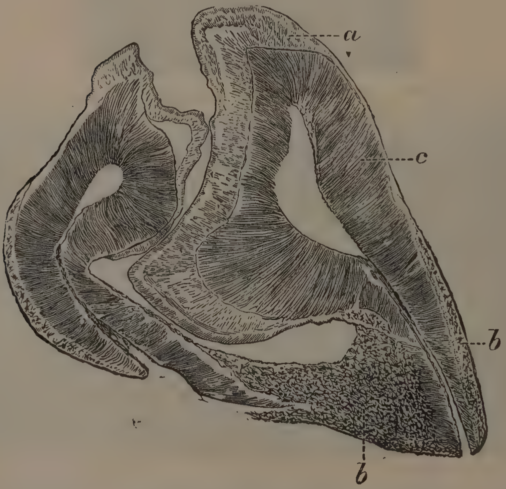


FIG. 766.—Section of fig. 765—*a*, enamel; *b*, cement; *c*, dentine. (From *Brit. Dent. Journ.*)

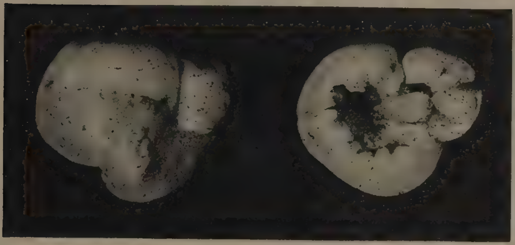


FIG. 767.—Dilated odontome from mandibular third molar region.

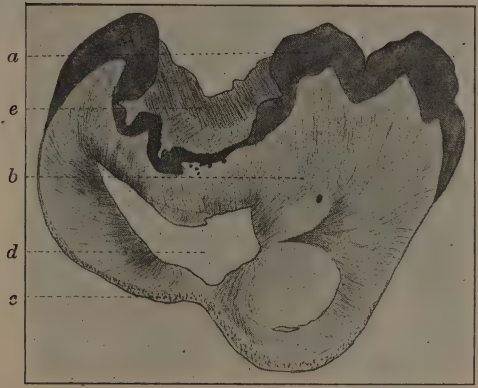


FIG. 768.—*a*, enamel ; *b*, dentine ; *c*, cement ; *d*, pulp cavity ; *e*, filling material. (Section of fig. 767.)

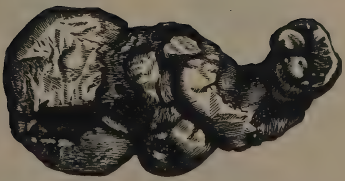


FIG. 769.—Radicular type of dilated odontome from maxilla of male, aged forty-five. (From *Trans. Odonto. Soc.*)

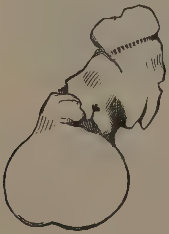


FIG. 770.
Radicular type of dilated odontome; the dilated portion consists of a confused mass of bone and dentine. (S. J. A. Salter.)

calcification; later[1] he has likened it to a benign neoplasm of limited growth such as a mole of the skin (a benign melanoma).

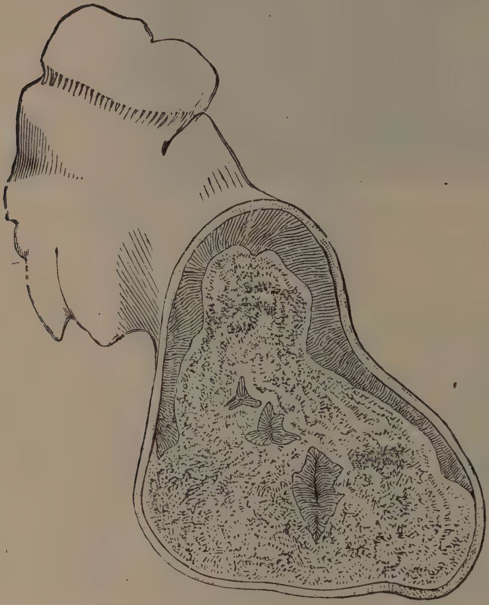


Fig. 771.

Radicular type of dilated odontome; the dilated portion consists of a confused mass of bone and dentine. (S. J. A. Salter.)

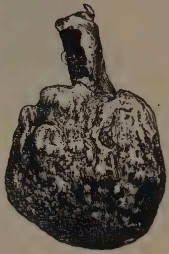


Fig. 772.—Radicular type of dilated odontome formed in connection with maxillary left third molar. (From *Brit. Dent. Journ.*)

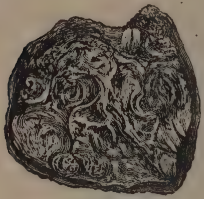


Fig. 773.—Section of fig. 772; it consisted largely of osteo-dentine, vaso-dentine and vaso-cementum. (From *Brit. Dent. Journ.*)

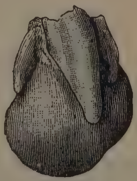


Fig. 774.

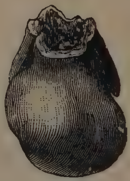


Fig. 775.

Figs. 774 and 775.—Dilated odontome in connection with a four-rooted maxillary molar. (From *Brit. Dent. Journ.*, 1896.)

It seems probable that some of these odontomes originate as an unco-ordinated attempt of the enamel organ to divide, as separate pulp canals are sometimes found (see figs. 757 and 763 to 768 inclusive) in addition to the dichotomous appearance; besides, with a better co-ordinated invagination, teeth with bifid crowns and the forms of incomplete unequal dichotomy are found in the same regions, and it is common both with these and dilated odontomes for the labial aspect of the crowns to simulate that of the normal tooth which the odontome represents or is near. If the invagination were a simple one with no attempt at dichotomy the attenuated pulp should entirely surround it, but it does not always do so, and there seem to be separate pulp canals.

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[1] *Brit. Dent. Journ.*, 1937, lxiii, 65.

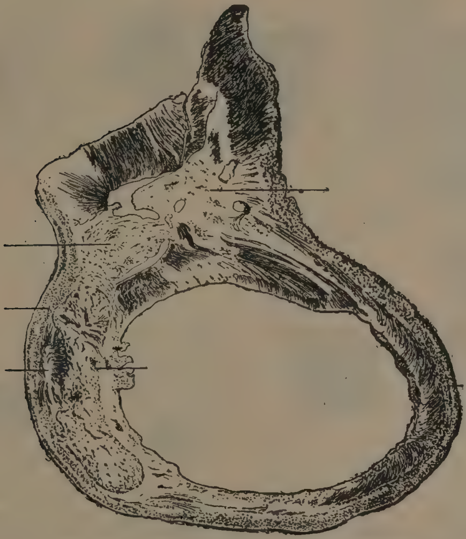

Fig. 776.—Section of specimen figured in 774 and 775.

It must be admitted, however, that other forms (figs. 758 to 762 inclusive) show but small signs of dichotomous growth, though there is plenty of evidence of unco-ordinated epithelial growth; in that case these forms should perhaps not be grouped with dichotomies but with the abnormalities of aberrant Adichotomous Growth, but a more exact knowledge of their development is needed before further subdivision of their classification can be usefully discussed.

Rushton suggests that the epithelial ingrowth in dilated odontomes is similar to that of complex odontomes, but that in the former the state of maturity of the tooth germ is more advanced before the error in growth commences ; he also suggests that the invagination occasionally originates from the sheath of Hertwig and diagrammatically summarises the simpler forms (fig. 777).

Origin.—When a tooth is absent from the normal series in the neighbourhood of one of these odontomes, as is usually the case, there can be little doubt that the odontome is due to aberrant growth of that tooth germ ; under other circumstances it can only be due to aberrant growth of a tooth germ in excess of the normal number, *i.e.* a complete dichotomy, or arise *de novo* from the dental lamina. The aberrant growth is sometimes more noticeable in those parts having origin from the dental papilla and tooth follicle, sometimes in those derived from the enamel organ, or, it may be, all of them.

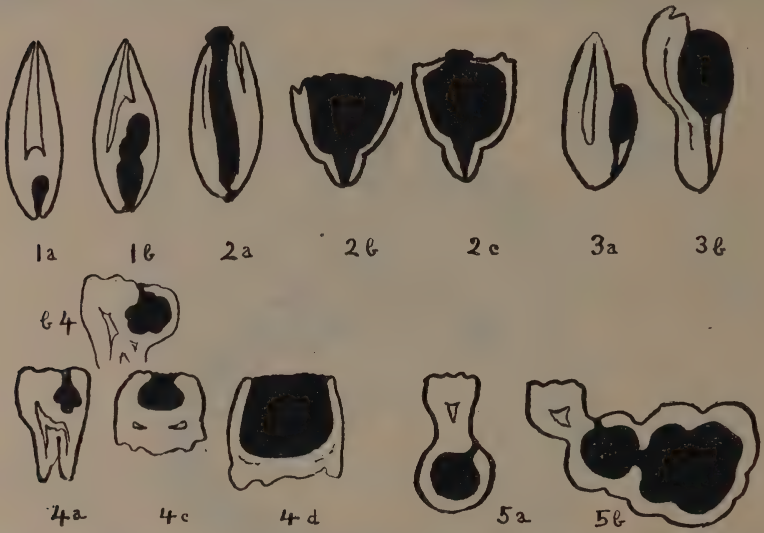

FIG. 777.—Diagrams of different types of dilated odontomes. 'The black area represents the area of a section taken up by the invaginating tissue, which may have become completely degenerate, or consist of connective tissue with epithelial remnants, or enamel, or bone. The upper row is represented as of maxillary teeth, the lower as of mandibular.' (Martin Rushton.) (From *Brit. Dent. Journ.*)

Etiology unknown ; the invaginating growth of the enamel organ producing the abnormality appears to be of a neoplastic nature.

Treatment.—If they give rise to untoward signs or symptoms, due either to their size, pressure on neighbouring parts, or infection (which they frequently do), or detract from appearance, they should be removed.

(iv) Unco-ordinated: Complex Odontomes (Compound when Multiple).—Of the complex odontomes originating in tooth germs of the normal series there are at least two and probably more varieties, in that some show unco-ordinated growth of part of the tooth germ only, as in fig. 778 and perhaps 785, while others appear to involve the whole of one or more tooth germs and to be entirely of neoplastic origin (fig. 779); other cases have been recorded, as that by Schmitz and Witzel[1] in which, not only were many tooth germs involved, but the separate abnormalities that each germ produced showed a familial tendency in that they occurred in two brothers; and still others in which the abnormalities were symmetrical, as in an early case recorded in the mandible by Oudet[2] in which each mass was apparently made up of the incomplete crowns of incisors and canines covered with enamel. It will be noticed that of the above examples three involved more than one abnormal production, and so may be grouped as compound odontomes, but complex and compound odontomes will be found to be closely related, indeed to the point of mergence.

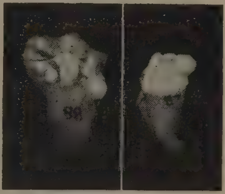

FIG. 778.—Compound odontome made up of two small complex odontomes, erupted in place of and representing mandibular left permanent first and second molars respectively. Female, aged 36. The rest of the dentition was normal. The crowns are unco-ordinated nodular masses, but the roots show some co-ordination. (× 1½.) (E. S.)

Generalising, complex odontomes consist of irregularly shaped nodular calcified masses composed of enamel, dentine, and cementum; usually they do not possess a single pulp chamber, but many irregularly directed canals containing pulp tissue from which dentinal tubes radiate; the enamel and dentine may be irregularly arranged (see figs. 779 to 784) or in definite alternate layers (see figs. 785 and 786). Nodules of enamel are often dotted about on the surface; usually they bear no external resemblance to a tooth; vascular channels are common and interglobular spaces are common in the dentine. They are commonest in the molar region and are most often noticed in early adult life, though sometimes in early childhood. Like teeth they tend to erupt, and it is often at that period that they are first noticed. Usually they give rise to no symptoms, unless on account of their size, till they commence to erupt, when infection may occur and inflammatory symptoms be noticed accompanied by the formation of fistulæ and discharge. In ungulates they have been known to erupt and come into occlusion (fig. 787), and a similar case

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[1] *Deuts. Monats. f. Zahn*, 1901, 19, 126.

[2] *Nouveau Journ. de Méd.*, Paris, 1821, 10, 245.

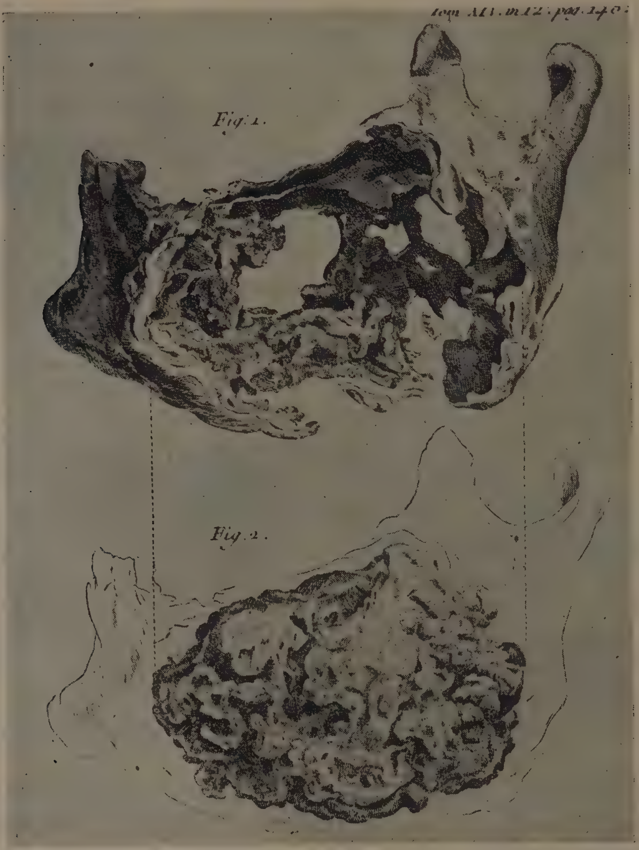


Fig. 779.—A large, wholly unco-ordinated complex odontome. Bordenave's (Morelot's) case. (From *Mém. de l'Acad. Roy. de Chir.*, Paris, 1774, 5, 352.)

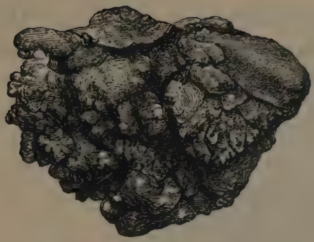


Fig. 780.—Complex odontome. (From 'Injuries and Diseases of the Jaws.' C. Heath.)

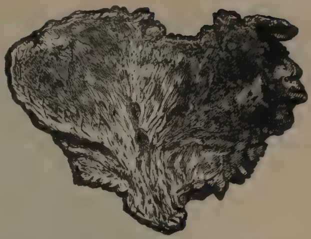


Fig. 781.—Section of odontome shown in fig. 780. Measured 1½″ × 1″ × 1¼″. Weighed 315 grs.

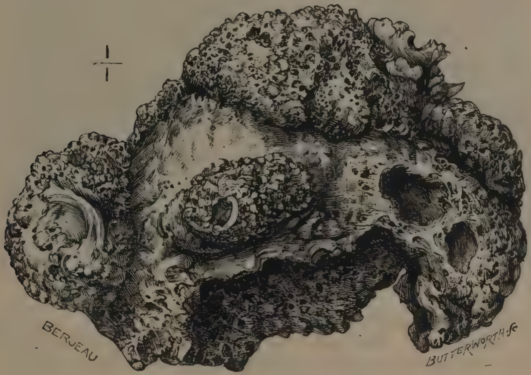


Fig. 782.—Complex odontome removed from Kaffir boy, aged fourteen, from left molar and premolar region, composed of imperfectly calcified dentine and cementum but no enamel. Measured 70 mm. × 62 mm. × 39 mm. Weighed 885 grs. (From 'Tumours, Innocent and Malignant.' J. Bland-Sutton.)

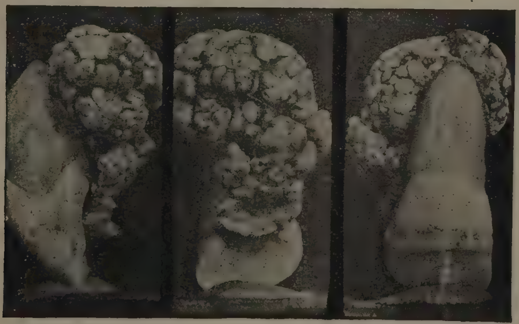
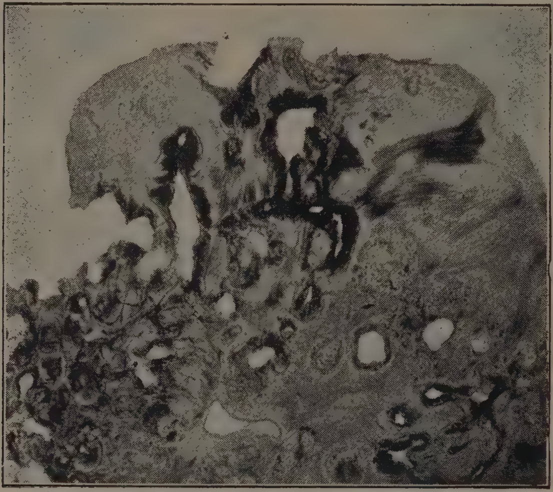


Fig. 783.—Complex odontome removed from $\underline{1|}$ region; the odontome was not in continuity with the tooth. Composed of enamel, cementum, and a few dentine tubes. (From *Trans. Odonto. Soc.*)

occurring in the maxilla of a horse was recorded by Broca.[1] In size they vary from that of a small nodule weighing a few grains to a mass weighing 885 grs. (see fig. 782). They are covered by a fibrous capsule. To the naked eye the constituents seem to vary, some seeming to have no enamel and others no cementum.



× 12.

Fig. 784.—Section of complex odontome with structure 'complex and very mixed,' composed of osteo-dentine mainly, but also enamel and a little cementum; there were many openings and canals.

(From *Trans. Odonto. Soc.*)

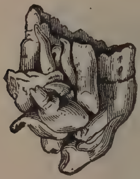


Fig. 785.—Complex odontome from first premolar region.

Occasionally they bear some resemblance to a number of denticles joined together with some additional unco-ordinated new growth (see fig. 785), and remind one of one type of imperfectly co-ordinated incomplete dichotomy (see fig. 751). Radiograms are an invaluable aid to diagnosis.

When these odontomes arise from tooth germs of the normal series it is rare to find more than one in the same mouth; such multiple productions, when they do occur, are called compound odontomes, and under these circumstances some of the productions seem only to be imperfectly co-ordinated rather than

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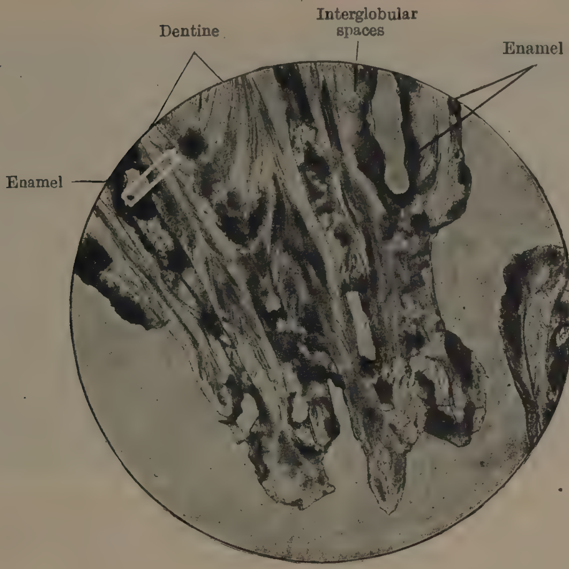
[1] 'Traité des Tumeurs,' Paris, 1869, ii, 320.



× 24.

FIG. 786.—Section of complex odontome with tissues arranged in alternate layers. (J. Lewin Payne.) (From *Brit. Dent. Journ.*, June 15, 1904.)

unco-ordinated in their growth, as in the case recorded by Schmitz and Witzel already referred to.

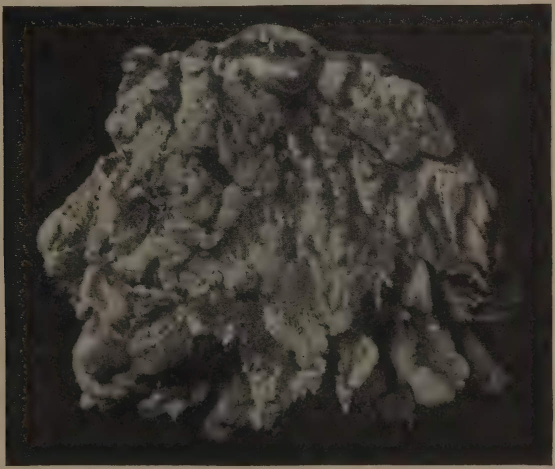


FIG. 787.—Complex odontome from the mandible of an Abyssinian buffalo (*Bos caffer æquinatialis.*)

Pathology.—The great majority of these odontomes appear to be the result of unco-ordinated growth, sometimes of part, but usually involving the whole of a tooth germ or germs, and in many cases they seem to be the result of neoplastic growth, primarily of the enamel organ. If a specimen of this type be sectioned while still in the developmental stage and *before any deposition of dentine matrix and calcification has taken place*, it will at once be seen that the growing columns of epithelium, originating apparently from the ameloblast layer, will simulate a basal-celled carcinoma or multilocular cyst, so that it would appear that some of these growths may originate as basal-celled carcinomata of fully formed enamel organs, and that their ultimate growth is limited either by their calcification, or by infection as they get big enough to reach the surface, possibly by the process of eruption.

Etiology.—There can be little doubt that most of these are neoplastic in origin; rarely is there a definite history of trauma, though this was so in the first recorded case (by Fauchard, in 1728),[1] the trauma occurring at the age of four years and the odontome later developing at that site, and it is noteworthy that basal-celled carcinomata have occurred at the site of trauma elsewhere in the body. The fact that there have been familial and symmetrical forms, however, seems to indicate that some are congenital.

Origin.—As, when these odontomes occur, a tooth is sometimes absent from the normal series, and occasionally more than one tooth, there can be but little doubt that they sometimes arise from aberrant growth of a tooth germ or germs of the normal series; this being so, there is every reason to believe that they may sometimes arise from aberrant growth of germs in excess of the normal number.

Treatment.—They should be removed.

Productions simulating Complex Odontomes.—These are definitely the result of trauma and the attendant inflammation and infection causing a general mix-up of the formative elements of a developing tooth, which, however, recover enough to continue their function. As far as we know, the degree of lack of co-ordination in their growth is that of the displacement and damage caused by the initial trauma and the consequent infection. They have been found in animals in such prominent teeth as may be exposed to trauma (figs. 788 to 792 inclusive) and

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[1] 'Le Chirurgien-dentiste,' Paris, 1728, 357.

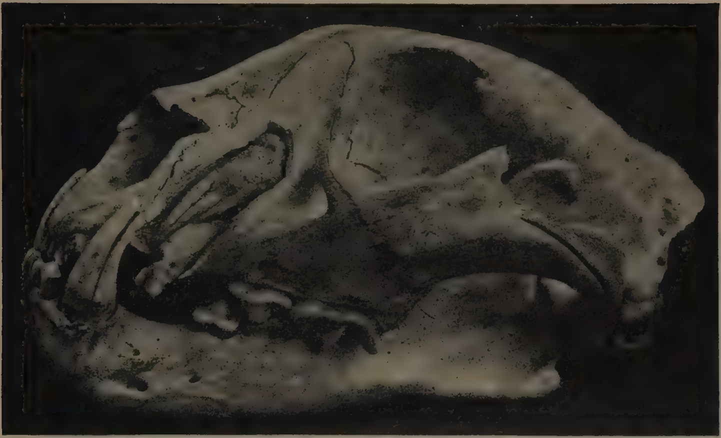

FIG. 788.—*Panthera pardus* (Leopard). R.C.S. Odonto. Section G, 63, 2. Showing result of severe traumatic injury (fracture) of maxillary left deciduous canine exposing the pulp, and consequent faulty development of permanent canine. (From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

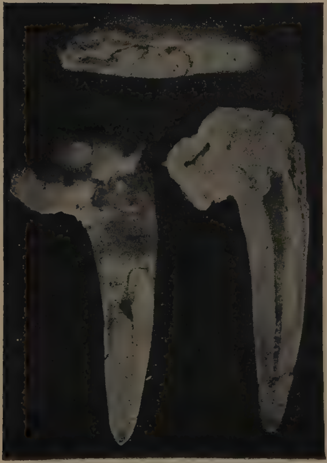

FIG. 789.—Section through maxillary left permanent canine of Leopard shown in fig. 788. (From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

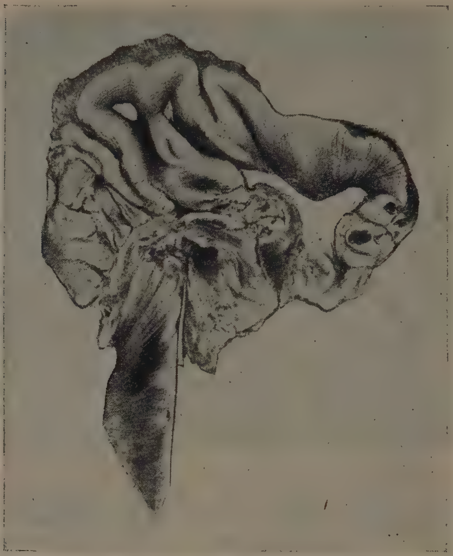

Fig. 790.—Section of part of permanent canine shown in fig. 789, showing disorganised formation. (From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

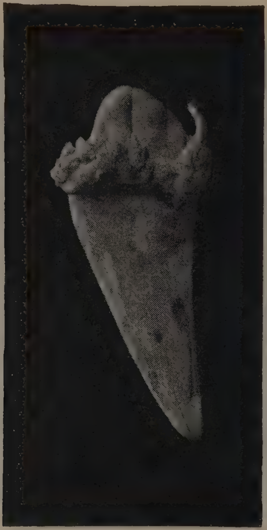

Fig. 791.—*Panthera leo* (Lion). R.C.S. Odonto. Section G, 63, 3. Showing result of injury to growing maxillary canine. (From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

occasionally occur in man (fig. 793). Unless the history is known it is conceivable that it might be impossible to differentiate between these and true complex odontomes. They differ only in degree from dilacerations (see p. 291) and such local abnormalities in structure as are described on p. 42.

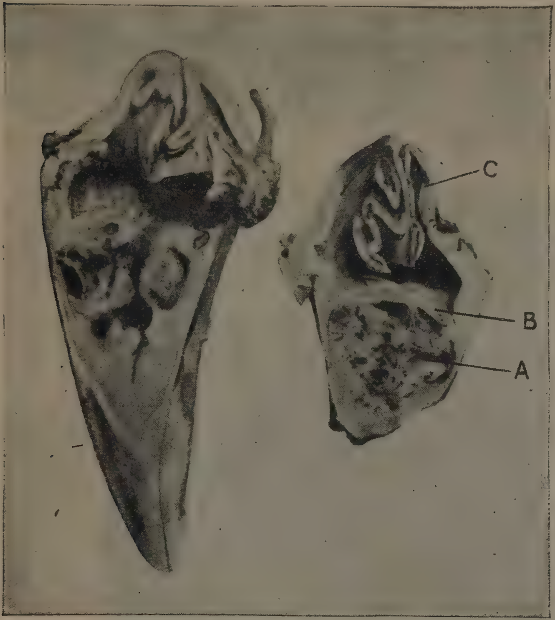

Fig. 792.—Section through injured canine shown in fig. 791, showing disorganised formation and masses of secondary dentine. (From 'Variations and Diseases of the Teeth of Animals.' J. F. C.)

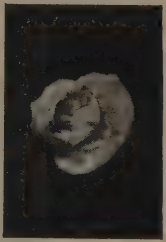

Fig. 793.—Mandibular right third molar subject to severe instrumental trauma and inflammation at age 10½, when impacted $\overline{7}|$ was removed, extracted at age $16\frac{3}{12}$. (E. S.)

(2) Arising from Tooth Germs in Excess of the Normal Series.

These are either complete dichotomies of germs of the normal series, or possibly may arise *de novo* from the dental lamina.

(i) **Co-ordinated.**—These consist of supernumerary teeth resembling those of the normal series and are *complete equal dichotomies*; sometimes they occur symmetrically, but sometimes do not. Occasionally a supernumerary tooth may be present on one side of the mouth and an extra cusp or cusps on one or more of the teeth in the corresponding region of the opposite side; and occasionally extra cusp formation is confined to that side on which the supernumerary tooth is situated. If there is room in the arch, supernumerary teeth may be in line with the other teeth; this is frequently so in the mandibular incisor and molar regions, but often they are crowded out. Supernumerary teeth are sometimes hereditary and occur more frequently in the maxilla. An excess in the number of teeth is slightly more common than a deficiency.

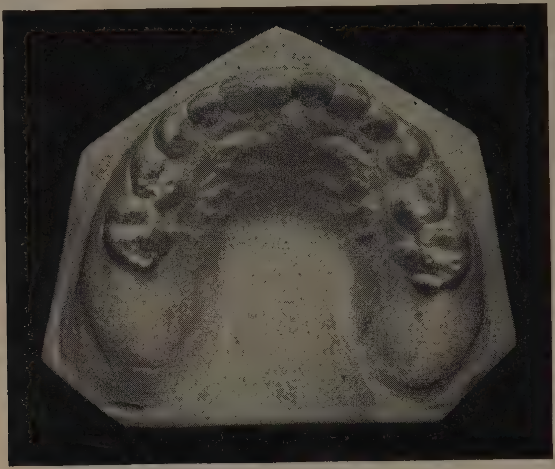

Fig. 794.—Symmetrical complete equal dichotomies in the maxillary incisor region, deciduous dentition. (H. Chapman.)

Taking all types of supernumerary teeth together, MacPhee [1] found twelve cases in 4,000 children aged five to twelve years, and Staphne has recorded a series of 180 cases in the maxillary incisor region, in twenty of which the condition was bilateral.

The co-ordinated type occurs in both the deciduous and permanent dentitions. The individuating action governing their growth is perfect in that they are invariably found in the same region of the mouth as the tooth which they simulate, and it is usually impossible to say which is the normal tooth and which is the tooth in excess of the normal.

Deciduous Dentition.—Excess teeth like those of the normal series are by no means rare in this dentition and have been recorded only in the maxillary and mandibular incisor regions[1]; they may or may not be followed by successional complete or incomplete dichotomies in the permanent dentition (fig. 794).

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[1] *Brit. Dent. Journ.*, 1935, lviii, 59.

Permanent Dentition.—They simulate most frequently maxillary and mandibular incisors, with less frequency the premolars and molars, and but rarely the mandibular canines; Salter has recorded the only case known[1] in which the maxillary canine was simulated in a manuscript note in the Royal College of Surgeons of England. In the maxillary incisor region they most frequently simulate the second incisor; in the premolar region as many as four supernumerary premolars have been found on one side; in the molar region, though they may exactly resemble a molar of the normal series, often they are only molariform in shape (figs. 795 to 797).

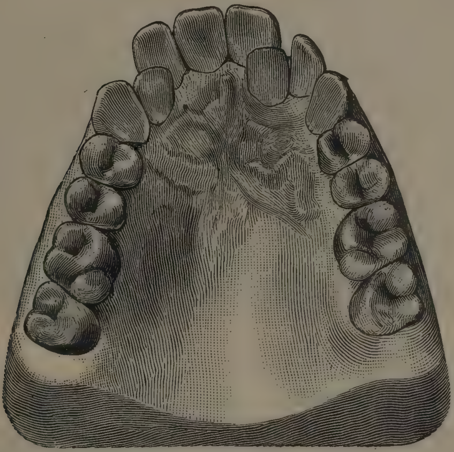

FIG. 795.—Symmetrical complete equal dichotomies in the maxillary incisor region, permanent dentition.

Supernumerary molariform teeth are commoner in the maxilla and are sometimes found symmetrically on the buccal aspect of the normal maxillary teeth in the angles between the second and third molars.

(ii) Imperfectly Co-ordinated.—These consist of supernumerary teeth unlike any of the normal series; apart from this, the same general remarks apply to them as apply to co-ordinated supernumerary teeth. They occur in two main varieties: Conical and Tuberculated. They are *complete unequal dichotomies.*

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[1] Since the above was written De Jonge Cohen has described a case of symmetrical duplication of the maxillary deciduous and permanent canines in the same individual. Proceedings of the Koninklinike Akademie van Wetenschappen te Amsterdam, xli, 2.

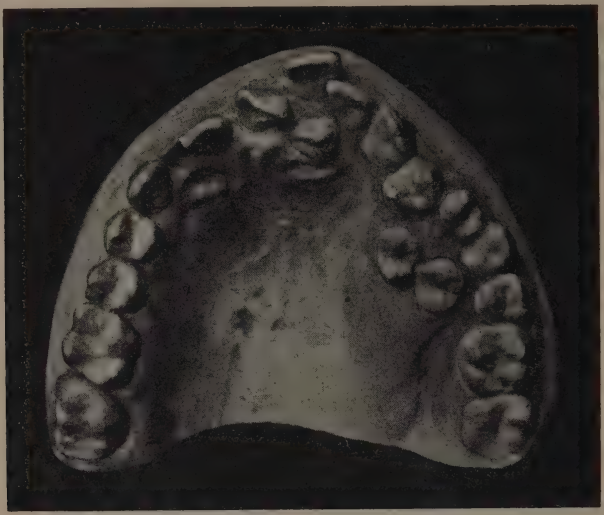

FIG. 796.—Model showing six premolar teeth on one side; there are, in addition, two other supernumerary teeth.

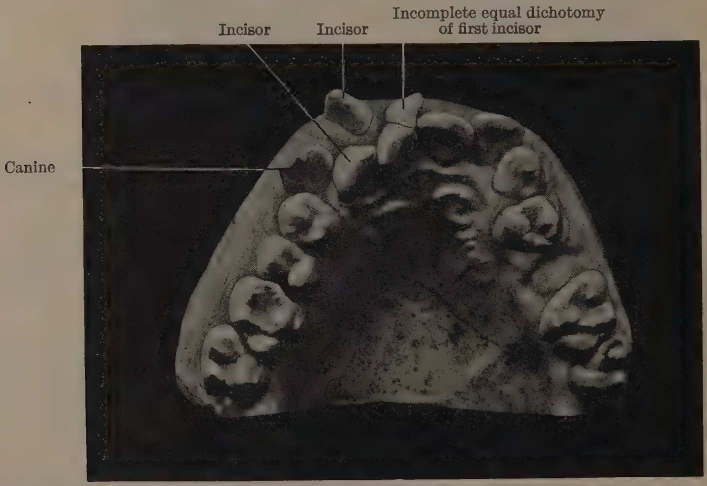

FIG. 797.—Model. A supernumerary incisor is present and the first incisor is tending to divide into two teeth.

Conical Complete Dichotomies. *Deciduous Dentition.*—Conical supernumerary teeth are not so common as those resembling teeth of the normal series; they occur in the incisor regions and may or may not be followed by complete or incomplete dichotomies in the permanent dentition (fig. 798).

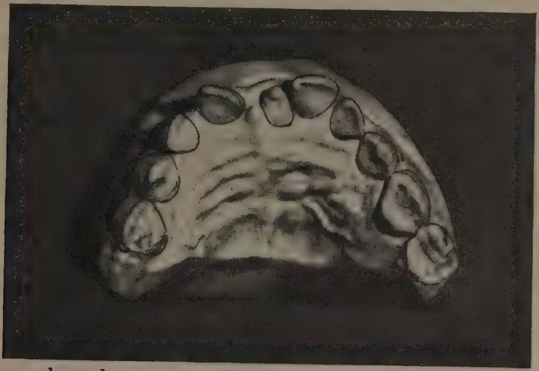

FIG. 798.—Cone-shaped supernumerary tooth situated between the maxillary deciduous first incisors.

Permanent Dentition.—Conical supernumeraries are by far the most common type of excess tooth occurring in this dentition; they are found most frequently in the maxillary incisor region, occasionally in the maxillary molar region and rarely in the mandible, and then usually posterior to the canine. They are usually asymmetrical, but this is not always so. Their roots are usually single, circular on section, straight and rather short,

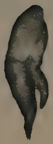

FIG. 799.—A conical shaped supernumerary tooth with two roots. (Museum of the Victoria Dental Hospital.)

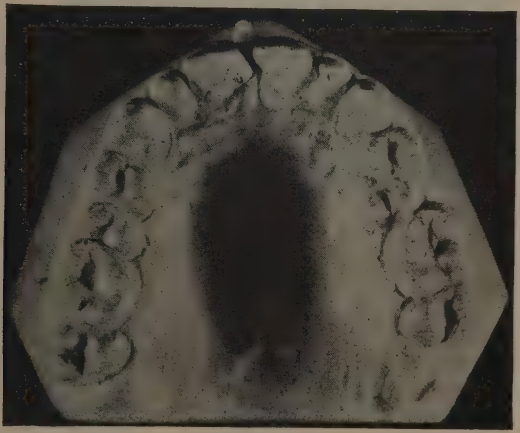

FIG. 800.—Conical-crowned tooth in excess of normal number between and labial to the maxillary permanent first incisors. (E. S.)

(From Bennett's 'The Science and Practice of Dental Surgery'—*Oxford Medical Publications.*)

but occasionally they are long and curved (figs. 799 to 803). An extra incisor is often associated with cases of cleft palate. In cases of cleft palate the usual arrangement from the mid-line is: first incisor, cleft, badly formed tooth, canine; the badly formed tooth is always immediately adjacent to and anterior to the canine and so has been termed the 'pre-canine,' but really is homologous with the incisor teeth. When a supernumerary tooth is present it is situated on the mesial side of the cleft and is often like one of the normal series, *i.e.* a maxillary second incisor (see figs. 804 and 805).

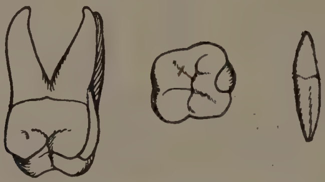
Fig. 801.—Maxillary molar and conical supernumerary tooth.

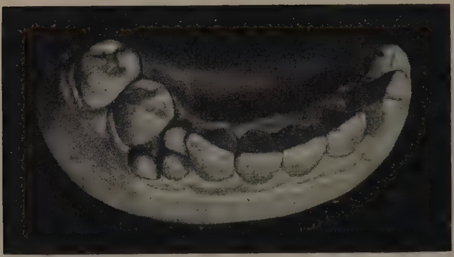
Fig. 802.—Mandibular canine replaced by three cone-shaped teeth. (Note reference to this figure on p. 751.)

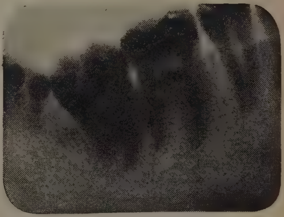
Fig. 803.—Imperfectly co-ordinated complete dichotomy in place of mandibular third molar, conical. (W. Peebles.)

Tuberculated Complete Dichotomies. *Deciduous Dentition.*—They have not so far been recorded in this dentition.

Permanent Dentition.—They occur, not infrequently, in the maxillary incisor region and are often symmetrical, their roots are usually single, circular on section and longer and stouter than are those of conical supernumerary teeth, though they may be slightly flattened or curved (fig. 806).

Treatment of Co-ordinated and Imperfectly Co-ordinated Complete Dichotomies.—When they are unsightly, functionless, or interfere with occlusion or normal masticatory movements they should be removed, and this is almost always desirable for the conical and tuberculated varieties; frequently it is desirable to remove

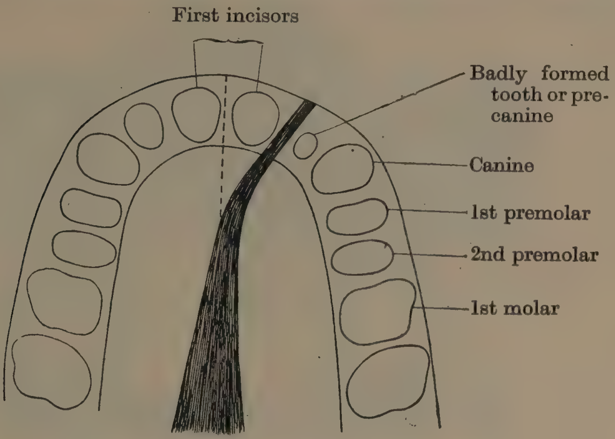

FIG. 804.—Usual arrangement.

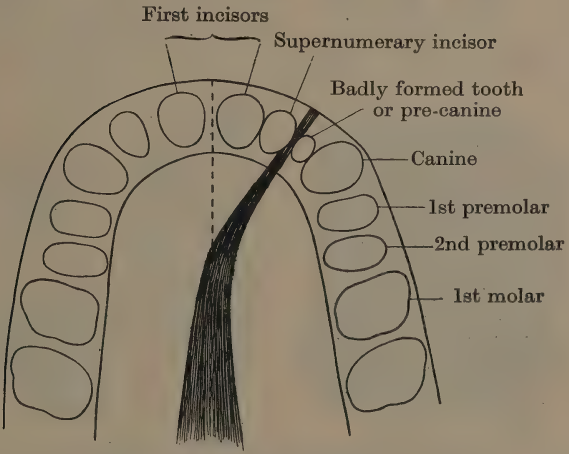

FIG. 805.—Unusual arrangement.

supernumerary teeth before they erupt as they are liable to cause displacement or crowding of the teeth of the normal series, particularly in the front of the mouth. Occasionally, when there is ample room, a supernumerary tooth like one of the normal series is a component part of the dental arch, is in good occlusion and should be left in place ; this occurs most often in the mandibular incisor region.

(iii) Partly Unco-ordinated. Some Dilated and some Complex

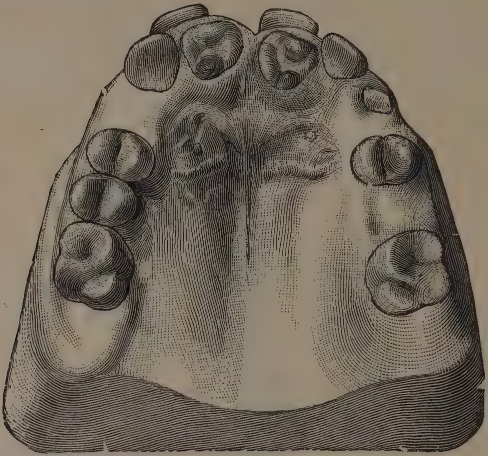

Fig. 806.—Symmetrical tuberculated supernumerary teeth posterior to the first incisors.

Odontomes.—These may arise from complete dichotomies, and except for that fact differ in no way from similar abnormalities of tooth germs of the normal series (see p. 726 *et seq.*).

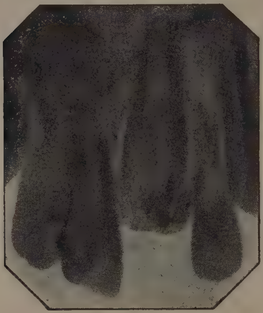

Fig. 807.—Radiogram of a compound odontome showing the odontome in position.

(A. H. Johnson.) (From *Proc. Roy. Soc. Med.*)

(iv) Unco-ordinated. Complex Odontomes.—When these are derived from dichotomous productions, except for that fact they differ in no way from similar abnormalities of tooth germs of the normal series (see p. 735 *et seq.*). A complex odontome is probably rare as a completely dichotomous production.

Compound Odontomes.—These odontomes far more frequently concern complete dichotomies than they do tooth germs of the normal series, and consist essentially of the production of a few to several hundred masses of calcified dental tissue ; these supernumerary productions may be of all types, from well-co-ordinated to unco-ordinated, but the latter type usually prevails (figs. 807 to 809); often each of these resembles a small supernumerary denticle of irregular shape, but they may consist of cementum, dentine, or dentine and enamel separately; occasionally these denticles erupt in small groups, when they are classed as supernumerary teeth (see fig. 802). Far more often these odontomes are first noticed as swellings, sometimes but not always cystic. Apparently in some cases the mass is made of cystic loculi, each containing one or several denticles, so that it

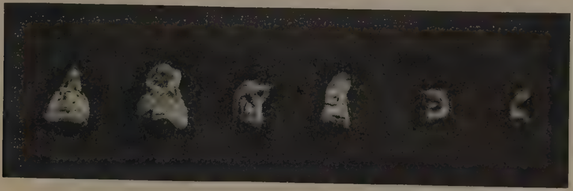

FIG. 808.—Denticles removed from the odontome shown in fig. 807. (A. H. Johnson.) (From *Proc. Roy. Soc. Med.*)

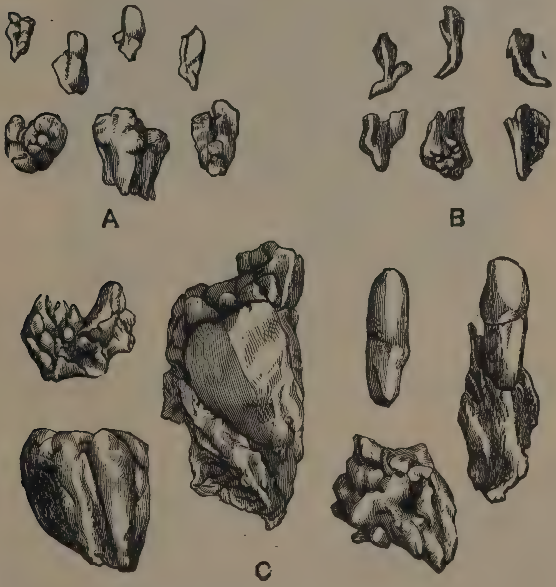

FIG. 809.—Denticles from three compound odontomes. (From *Trans. Odonto. Soc.*)

appears as if a number of small dentigerous cysts composed the whole; it is probable that these cysts originate as cysts of eruption, and coalesce owing to their crowded proximity. They have also been recorded in ungulates (fig. 810). They are first noticed in youth or early adult life and give rise to slowly growing painless swellings which occasionally suppurate. One or more teeth may be absent from the normal series, but this is not always so, and occasionally the odontome may interfere with the eruption of a tooth of the normal series (see fig. 811); in such cases if the denticles are removed at somewhere near the time when the eruption of the obstructed normal tooth is due, this tooth may then erupt into its normal position in the arch as in the case of dentigerous cysts (see figs. 704 to 710 inclusive), but occasionally an orthodontic appliance may be needed to help it there as in a case recorded by K. C. Smyth.[1]

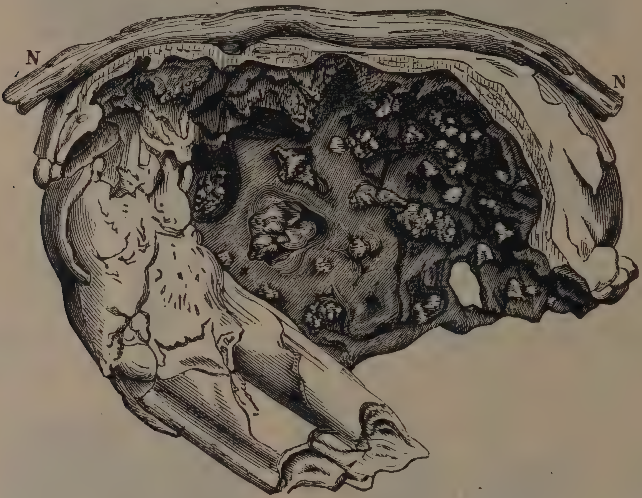

FIG. 810.—Section through the maxilla of a thar, showing compound odontome. N, the maxillary division of the fifth nerve. (Bland-Sutton.)
(From *Trans. Odonto. Soc.*)

These odontomes nearly always arise as dichotomies from or coincident with the permanent dentition, but Axel[2] has recorded one in a boy aged four years which contained twenty-four imperfectly co-ordinated denticles and apparently originated from or with the deciduous dentition. Radiograms are an invaluable aid to diagnosis.

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[1] *Proc. Roy. Soc. Med.*, 1933, xxvi, 1561.
[2] *Journ. Amer. Dent. Assoc.*, 1937, 24, 457.

Etiology.—When a tooth or teeth are absent from the normal series there can be little doubt that these odontomes are produced by unco-ordinated dichotomous growth of the germs which should have produced them. It is possible that they may also be produced by exuberant growth from the dental lamina giving rise to supernumerary denticles. These denticles may or may not become involved in cysts which they may give rise to if they are unable to erupt. It has been suggested that they may arise from non-union of the denticles of a compound tooth, but they often exceed these in number.

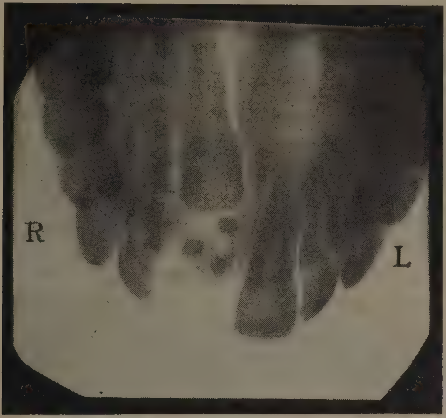

Fig. 811.—Radiogram of a compound odontome which contained nine denticles it is preventing the eruption of the maxillary permanent first incisor. Male, aet. 11 years. (H. D. Clapham.)

Treatment.—They should be removed, but if they hinder the eruption of a tooth of the normal series and are treated some time near the date of the normal eruption of that tooth, that tooth should not be removed, as when the obstruction to its eruption is removed it may erupt into its normal position or be helped there by an orthodontic appliance.

Note.—In order to emphasise the close affinity and occasional coincidence of different forms of odontome, let us imagine that two or more supernumerary denticles, of normal shape or otherwise, develop adjacent to each other: (i) If they erupt they are called *supernumerary teeth.* (ii) If, while unerupted, cystic formation takes place over and around them separately, they are called *dentigerous cysts.* (iii) If the cysts coalesce around them, that is called a *compound odontome.* (iv) If these denticles are united, either developmentally or pathologically, that may be called a *complex odontome.*

Origin and comparison of all incomplete and complete dichotomies, including complex and compound odontomes.—See also Note on p. 753. There have been many hypotheses put forward to account for one or other of the above. Whatever the explanation in any particular case may be, it must depend primarily on aberrant growth originating from the epithelial dental formative organs, such growth being always of an exuberant nature; in the case of extra roots it is also true because their ultimate form is outlined primarily by the epithelial sheath of Hertwig.

Extra cusps, extra roots, enamel nodules, and dichotomies of the whole tooth germ whether complete or incomplete are all apt to appear at definite and very constant sites, and in a series of specimens could be shown to merge from the first mentioned to the last, so that no definite line of demarcation could be drawn between each; the fact that these variations are sometimes symmetrical assists in upholding this view, because occasionally a supernumerary tooth on one side of the mouth is associated with extra cusps on the other side.

All are occasioned by a dichotomy incomplete or complete from the epithelial dental formative organs of a tooth of the normal series, but in the case of a separate entity such as a supernumerary tooth it may also be an aberrant development direct from the dental lamina. Similarly, no hard and fast line can be drawn between supernumerary teeth and some compound composite odontomes, and the developmental joining of the denticles in this latter would make it identical with some forms of complex odontome. It is therefore probable that all these variations are merely differences in degree of the same thing, *i.e.* exuberant growth having origin in the epithelial dental formative organs, most often of a tooth of the normal series, but occasionally possibly of the dental lamina.

Teeth in excess have been explained as atavisms or throwbacks to ancestors having larger dentitions, but though this may be true with regard to the maxillary incisor region it does not explain the occurrence of six premolar teeth on one side of the maxilla. It has also been said that they occur in the neighbourhood of developmental fissures; this is sometimes true, but it is also true that they are not infrequent elsewhere.

Bolk explains extra cusps, incomplete dichotomies of the whole tooth germ, and complex teeth as being caused by coalescence of a tooth germ of the normal series with another of an ancestral dentition, and Kukenthal thought that complex teeth were derived in this manner. On the other hand, Marett Tims believed that complex teeth were caused by coalescence of adjacent tooth germs of the same dentition brought about by shortening of the jaws. Tomes showed that the coalescence of rudimentary tooth germs might form a continuous sheathing mass of dental tissues, exemplifying this in the lizard Sphenodon. It is possible that some of these variations may be of the nature of evolutionary throw-forwards,[1] and one may conclude the causal origin of these variations is uncertain and may not be the same in all cases.

(C) CONNECTIVE TISSUE PRODUCTIONS.

(1) Fibrous 'Odontomes.'—These are swellings caused by hyperplasia of the fibrous connective tissue of the tooth follicle which is much increased in amount and arranged concentrically around a tooth or in association with it; occasionally ossification may occur in the connective tissue. They have occurred in ungulates, carnivores, and marsupials as well as man and are said to occur only in rickety subjects; unless therefore some can be shown to originate as neoplasms of the tooth follicle none should be classed as odontomes. It may be difficult to differentiate between the hyperplasic connective tissue of these 'odontomes' and that of endosteal fibromata or neuro-fibromata (figs. 812 and 813).

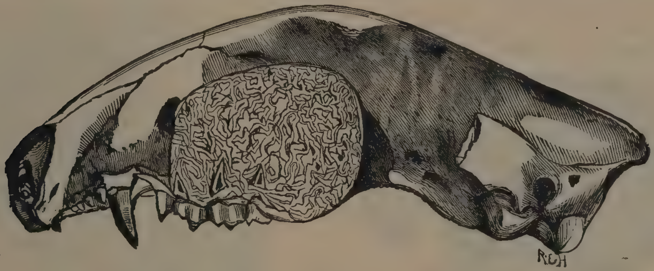

FIG. 812.—Portion of the skull of a dasyure, showing a fibrous 'odontome' in section. The tumour was intimately connected with a molar tooth. (Bland-Sutton.) (From *Trans. Odonto. Soc.*)

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), 1922, xv, 72. (E. S.)

Etiology.—Rickets, in so far as is known.

Treatment.—If they give rise to untoward signs or symptoms they should be removed.

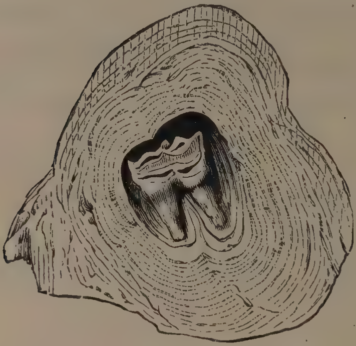

FIG. 813. (From *Trans. Odonto. Soc.*)

(2) **Cementomata.**—These are swellings composed entirely of cementum and usually occur attached to the roots of teeth; one variety appears to be commoner in ungulates (horses) than in man (fig. 814).

Some of them appear to be of the nature of true neoplasms, and in these the tissue composing them is described as being non-laminated and very vascular, but though they are firmly attached to teeth it is by no means certain that the tissue composing them is cementum. The structure of bone may on occasion be so similar to that of cementum that it is sometimes difficult if not impossible to differentiate between them, and it is extremely likely that many of these odontomes are of the nature of bone rather than cementum (figs. 815 to 820). They may arise through calcification taking place in a fibrous odontome. Their surface is usually rough and covered with numerous small nodules.

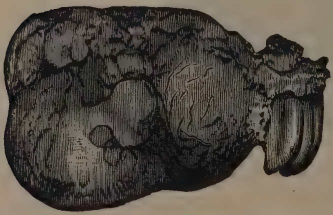

FIG. 814.—Cementome. (Forget's case.)

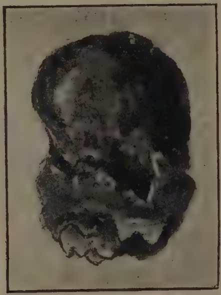

FIG. 815.—Odontome composed of tissue resembling cancellous bone. (J. H. Mummery and A. T. Pitts.) (From *Brit. Dent. Journ.*)

Others, and these are by far the commoner, are inflammatory in origin (see Chapter XV), and range from the very common result of a chronic periodontitis of low intensity to a lobulated mass of cementum of some size; occasionally two or more teeth may be joined together by this means, pathological union

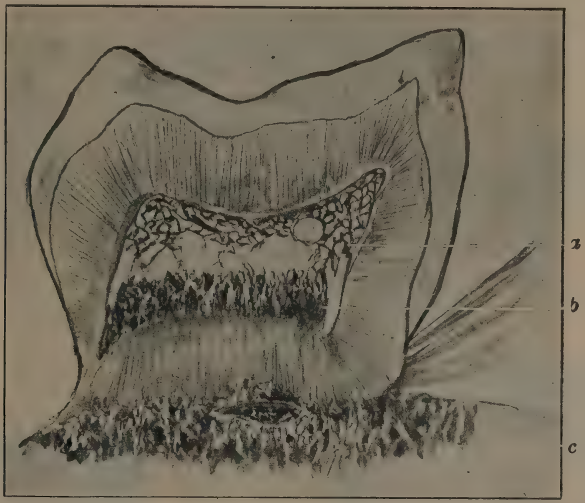

Fig. 816.—Section through the pulp chamber of part of fig. 815; (*a*) degenerated pulp tissue; (*b*) bony tissue at base of the pulp chamber continuous with the pulp; (*c*) cancellous bone of odontome.
(J. H. Mummery and A. T. Pitts.) (From *Brit. Dent. Journ.*)

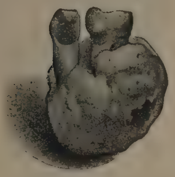

Fig. 817.

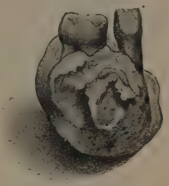

Fig. 818.

Odontome composed of tissue resembling bone rather than cementum. (From Wedl's 'Atlas of Pathology of the Teeth.')

being produced in this manner; pathological union is so far unknown between deciduous teeth, the reason being the relatively brief period for which they are present in the mouth and the fact that their roots become absorbed and disappear. Sometimes, again, when a tooth such as the maxillary third molar is misplaced, or lack of bony growth does not allow it to rotate into its normal position, it may in its growth and efforts to erupt engage with the roots of the second molar, and the resistance to its eruption result in excess production of cementum, so that they become joined by this tissue (fig. 821); occasionally this occurs symmetrically as in fig. 822, and very rarely there may be so little room that neither the second nor third molars have room to come into alignment and erupt, and they all become joined in

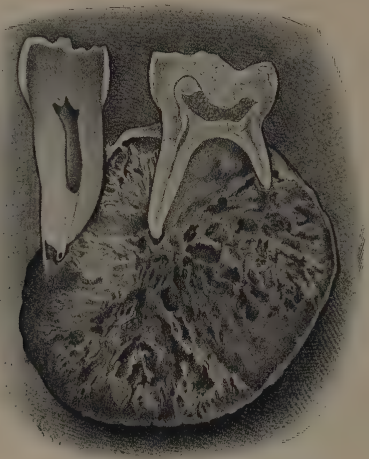

FIG. 819.—Section of specimen in figs. 817 and 818. (From Wedl's 'Atlas of Pathology of the Teeth.')

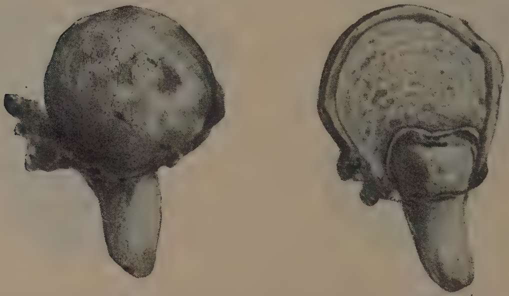

FIG. 820.—Odontome covering the crown of a mandibular molar (half the odontome has been removed to show its relation to the tooth). Structurally it was composed of cancellous bone. (D. P. Gabell.)

positions comparable to those which they occupy during development (fig. 823). Similarly, excess production of cement is often found on the roots of unerupted misplaced teeth such as maxillary canines, being probably produced by the irritation occasioned by the resistance to the physiological efforts to erupt. The masses of calcified tissue produced under these latter conditions are true cementum, laminated, non-vascular, showing a variable number of lacunæ and canaliculi and not in continuity with bone.

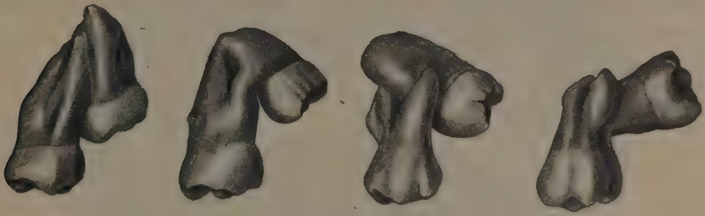

FIG. 821.—Pathological union.

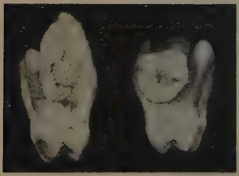

FIG. 822. — Symmetrical pathological union of maxillary second and third molars. (J. Noriskin.)

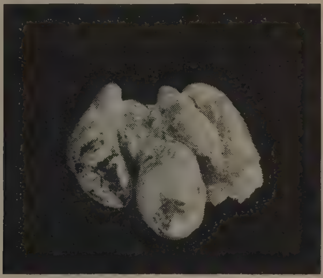

FIG. 823.—Pathological union of maxillary right permanent first, second and third molars; only the first had erupted, insufficient room (growth) preventing the others, so that the roots became joined by cemental hyperplasia. (A. C. King.)

They give rise to no signs or symptoms save such as may be occasioned by increase in size, pressure on neighbouring parts, and infection.

Treatment.—If they occasion untoward signs or symptoms or maintain infection they should be removed.

Malignant Odontomes.—No certain case of a malignant odontome is known, though several cases [1] go far to suggest that they probably have occurred of a sarcomatous nature (figs. 824

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[1] See 'Report on Odontomes,' 1914.

and 825). It is probable that some cases described in the past as being spheroidal or columnar celled carcinomata were really tumours of the same nature as the multilocular cyst. It is quite possible that true carcinomata might have origin from any of the remnants of the epithelial dental formative organs, but it has not yet been proved. Cells containing melanin have been found in a developing complex odontome, and though this is suggestive of possible malignancy the particular case in which it occurred was not of that nature.

Teratomata.—There is the probability that some odontomes, notably of the complex and compound composite types, and some cases of multiple supernumerary teeth may be of the nature of teratomata and consist of partial reduplication of parts; such a case is one recorded by J. H. Gibbs.[1] In this, a child aged two years and nine months presented a swelling on the right side of the mandible. All the deciduous teeth were fully erupted: projecting through the gum below the level of the deciduous canine was a tooth composed of the geminated crowns of a deciduous second incisor and canine. The swelling was opened and six other teeth found (see figs. 826 and 827). These teeth proved to be four deciduous molars and two permanent molars,

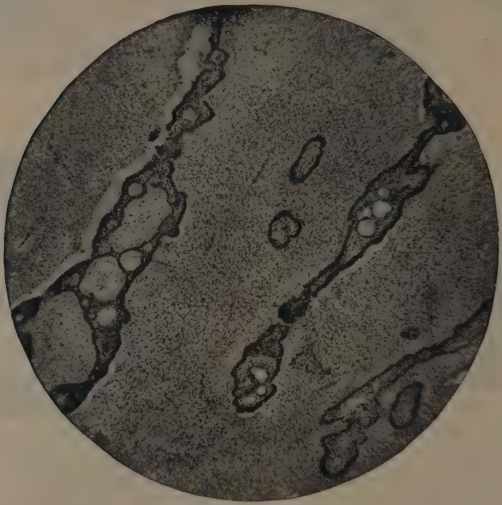

FIG. 824.—(Low power.) Composite embryoplastic odontome showing strands of enamel epithelium lying in a sarcomatous ground substance. The strands enclose small cysts and areas of stroma. (Eve's case.)

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[1] *Trans. 17th Int. Med. Cong.* (Stomat. Sect.), London, 1913, 19.

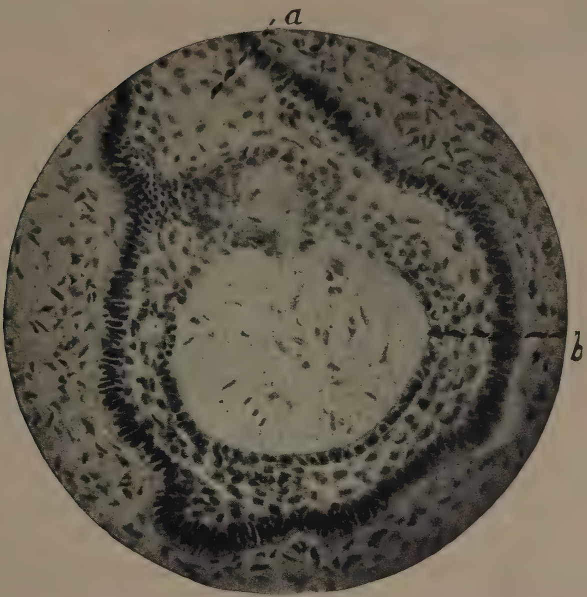

FIG. 825.—Part of section shown in fig. 824, but under a higher objective. The figure shows a rounded strand of enamel epithelium, lined externally with columnar cells. The intermediate layer exhibits a reticulum (*a*). At its centre the strand is penetrated by a round area of stroma (*b*).

three of which (two deciduous molars and one permanent molar) belonged morphologically to the opposite side. In this case not

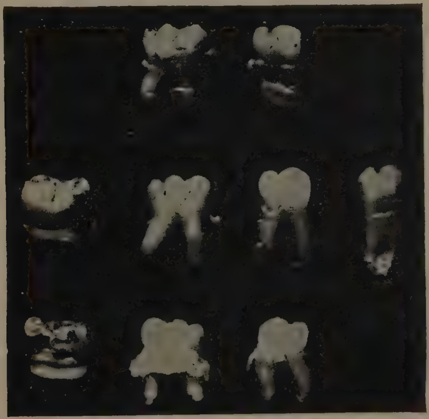

FIG. 826.—*Top row*—in use; *middle row*—geminated second incisor and canine, two deciduous molars and permanent molar; *bottom row*—two deciduous molars and permanent molar morphologically belonging to left side. (From *Trans. 17th Int. Med. Congress* (Stomat. Sect.), London, 1913, 19.)

only teeth but also part of the alveolar portion of the mandible was apparently reduplicated.

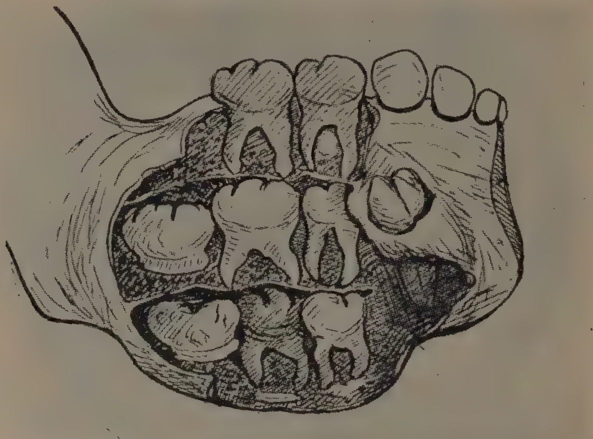

Fig. 827.—Drawing of the tumour made after the operation to show the relative position of the parts. (From *Trans. 17th Int. Med. Congress* (Stomat. Sect.), London, 1913, 19.)

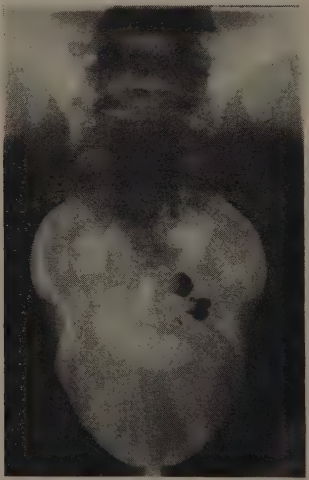

Fig. 828.—Radiogram showing two partly formed molars in an ovarian teratoma. (E. S.)

Apart from the jaw, teratomata containing teeth occur in ovarian teratomata (fig. 828) and very rarely in pituitary, testicular and pineal teratomata.

CHAPTER XXIII

The Treatment of Dental Disease in Children.

In connection with the treatment of dental disease in children there are a few points which may conveniently be considered under a separate heading. No part of the work of the dental surgeon requires more tact than the treatment of children. Some possess the gift of readily winning the confidence of the young, but this power is not given to all. The operator must constantly bear in mind that he is dealing with children, and that they are interested only in simple topics. Every effort should be made to attract their attention and to avoid giving them pain; but if pain is unavoidable, in no circumstances should they be misled or deceived.

Too great a stress cannot be laid upon the importance of regular and thorough attention to the teeth of children. Dental supervision should commence as soon as the deciduous dentition is complete, and the child should be brought to the dental surgeon at least three times a year for examination. Cavities can then be filled while quite small, with the results that the teeth are more easily saved and the operation is rendered practically painless—a point of no small importance in dealing with children.

Fortunately the teeth of children up to the age of ten or eleven years are nothing like so sensitive as are those of adults. Children under six years of age only rarely complain of pain from caries of the deciduous teeth even when the pulp is exposed; 'toothache' in their case almost invariably indicates suppuration, usually an alveolar abscess.

In treatment of dental disease in children **the importance of rendering the mouth functional** must be clearly kept in mind. If the mouth is rendered functional it will be kept clean by natural means. The chief causes of a functionless mouth in children are :—

(*a*) Mouth-breathing.
(*b*) Dental Caries.

(*a*) **Mouth-breathing** arising from nasal obstruction produces a persistent gingivitis of the gum in the front of the mouth, and predisposes the teeth to caries and parodontal disease, especially the incisor teeth. Mouth-breathing is often overlooked, more particularly in children who suffer from intermittent nasal obstruction. *The sign of nasal obstruction which may be regarded as diagnostic consists in a marginal gingivitis limited to the incisor teeth, the gums at the back of the mouth being healthy.*

Too much importance cannot be attached to the proper performance of nasal breathing, and, if there is sufficient nasopharyngeal trouble present to cause even intermittent mouth-breathing, the trouble must be removed. *Unless a child regularly breathes through the nose it cannot have a healthy mouth.*

Nasal breathing is not necessarily re-established by the removal of nasal obstruction, and it may be necessary for an oral screen to be worn at night for some time till natural nasal functioning is fully established. Once the habit of mouth-breathing has been acquired, even though nasal obstruction be removed and correct breathing is performed by day, there is frequently a reversion to the bad habit during sleep throughout the remainder of life, with an accompanying and progressive anterior gingivitis (see pp. 253 and 507).

(*b*) **Caries of the Teeth.**—It is a matter of common experience that children with carious teeth ‘ bolt their food,’ thus suspending the function of mastication. *Unless mastication is properly performed the mouth cannot be kept healthy.* It is essential, therefore, that children's teeth should be rendered capable of performing their function without the least discomfort.

Small cavities, whether in the crown or approximal surfaces, should be filled with amalgam, and special care should be taken to restore the contour of the tooth. Approximal cavities, when they have been opened up and the caries removed, are usually cup-shaped, and the retentive shaping must therefore be carried out by grooving. If the tooth is sensitive, it is sometimes judicious to bring the filling of amalgam against the approximal tooth. With children it is sometimes impossible to remove all the carious dentine from the deeper parts of the cavity, and in such cases the surface of the dentine should be covered with a little powdered nitrate of silver before the filling is inserted.

If the pulp of a tooth is exposed, the better line of treatment, in the majority of cases, is to remove the tooth. This practice may not coincide with that of some practitioners, but the impossibility of satisfactorily treating pulp chambers in children, and the fact that suppuration frequently occurs after treatment, inclines one to the opinion that, by extraction, the mouth is more likely to be rendered functional. The only instances where conservative treatment is indicated are cases of deciduous second molars in children under the age of six years—that is, where the permanent first molars have not erupted ; but even in these cases the teeth should be carefully watched, and, if periodontitis appears, they should be removed.

If it be decided to retain the tooth, the pulp should be removed from the coronal portion of the pulp cavity, and, if possible, from the root canals. Sterilisation should then be carried out and the pulp chamber filled with an antiseptic. No attempt should be made to fill the canals, the cavity in the tooth being completed with amalgam.

In a child a tooth with a septic pulp chamber often causes no objective symptoms, but yet is the seat of a slight chronic periodontitis ; moreover, the constant absorption of the septic matter from the pulp chamber is liable to cause chronic lymphadenitis.

The cases, however, which demand most serious consideration are those where the majority of the deciduous molars are carious and absolutely unsavable and the child suffers from the effects of toxic poisoning. If the permanent first molars are in position, the best treatment seems to be to remove all the deciduous molars with exposed or dead pulps. This treatment at once removes all sources of sepsis—a point of the greatest importance.

An objection has been raised that this method of treatment robs the child of masticating power. The answer to this objection is that such teeth are usually tender and avoided in the process of mastication, and that where many carious teeth are present the child 'bolts' its food instead of masticating it. In many such cases the lack of function of the permanent first molars can be easily demonstrated by the amount of debris which is present around these teeth. The removal of the carious deciduous teeth allows mastication to be efficiently performed and the masticating power is increased rather than diminished. Another argument advanced against this treatment is that the removal of the deciduous molars allows the permanent first molars to come forward and so cause, in the future, crowding of the anterior teeth. With regard to this argument, it may be pointed out that such travelling forward of the permanent teeth occurs mainly in mouths in which the growth of the jaws is hindered by want of function arising from insufficient mastication or from lack of nasal breathing. From observation—but the point is one on which final judgment is reserved—one is inclined to think that if by removing the deciduous molars the permanent first molars can be rendered functional, the growth of the jaw will be stimulated, and room will be made for the development of the second and third molars. With no forward pressure from the second and third molars, and with the first molars occluding correctly, there will be little, if any, forward movement. But, granting that the suggested treatment by extraction does cause a forward movement and subsequent crowding, the removal of four premolars will readily alleviate the condition, or, should circumstances make it seem desirable, a plate may be inserted to keep back the permanent first molars, but dentures or any other mechanical means should be avoided if possible, as such means can but interfere with the physiological uses of the tongue and teeth. A decision has to be made between the disadvantage arising from the possible loss of four teeth, on the one hand, and the constant presence of oral sepsis and all the sequelæ on the other. In our opinion the risk of sepsis is by far the greater evil. Still further, it must always be remembered that even if the deciduous molars are retained with the object of preventing a forward movement of the molars, it is quite possible that the removal of teeth to prevent crowding would be necessary, as in mouths such as those in question some interference with the development of the jaws would probably have taken place.

Cases are frequently seen in which caries has progressed rapidly in the permanent first molars to the extent of involving the pulp cavities before the formation of the roots is complete. Such teeth should be removed as they cannot be satisfactorily treated by conservative operations and, if left untreated, they become the foci of infection.

Accounts of a few cases will serve to illustrate the value of this method of treatment where a child is suffering from dental sepsis.

A girl, aged four years six months, when first seen was suffering from gastro-intestinal irritation. The teeth removed were: $\begin{array}{r|l} \text{d c b a} & \text{a b c d} \\ \hline \text{e d} & \text{d e} \end{array}$. The $\begin{array}{r|l} \text{e} & \text{e} \\ \hline \end{array}$ were not carious and were retained until the $\underline{6 \mid 6}$ erupted. The gastro-intestinal trouble cleared up and within four months the child increased in weight from 2 st. 4 lb. to 2 st. 8 lb. The normal weight should have been about 2 st. 8 lb., and the normal increase in four months about 1½ lb.

The following case also illustrates the marked improvement in weight which follows the removal of septic teeth : A boy, aged six years six months, was suffering from marked oral sepsis, enlarged glands in the neck, and gastro-intestinal irritation. He was weak and debilitated in appearance. He weighed 2 st. 12 lb. The following teeth were removed : $\frac{d\ \ b\ a \mid a\ b\ \ d}{e\ d\ b\ a \mid a\ b\ d\ e}$. The permanent first molars had just pierced the gum, and the $\overline{e \mid e}$, the pulps of which were not exposed or septic, were retained until the $\underline{6 \mid 6}$ had completely erupted. He was seen again seven weeks later, when his weight was 3 st. 3½ lb., or a gain of 5½ lb. in about seven weeks, the glands of the neck were considerably smaller, and his appetite and general health had increasingly improved.

A boy, aged seven years four months, stated always to have indigestion. The following teeth were removed : $\frac{6\ e\ d\ c \mid c\ d\ e\ 6}{6\ e\ d\ c \mid c\ d\ e\ 6}$. His weight was 3 st. 2½ lb. Two months later his weight had increased by 2½ lb. to 3 st. 5 lb., and all symptoms of gastro-intestinal trouble had ceased. The average weight of a boy of this age would be about 3 st. 9½ lb., and the normal increase in two months would be only a little under 1 lb.

Increase in weight after extraction does not always occur, and in a few cases there is a slight reduction in weight, but this is frequently traceable to the presence of sepsis in other parts—for example, in the tonsils or the naso-pharynx, as shown by the following case :—

A child, aged seven years six months, suffering from defective teeth, tonsils, and adenoids, was seen on February 23, when the following teeth were removed : $\frac{6\ e\ d\ c \mid c\ d\ e\ 6}{6\ e\ d\ \ \ \mid \ \ d\ e\ 6}$. The tonsils and adenoids were removed on May 3. The child's weights are given below :

	st.	lb.	oz.
February 23	3	10	8
March 24	3	9	12
May 25	3	9	12
July 26	3	10	12
October 15	4	0	6

In this case the improvement in weight and general health did not follow extraction until other sources of sepsis had been removed.

The cases quoted afford sufficient evidence that the loss of masticating power does not interfere with the general health of a child, but increases its power of metabolism by removing a source of toxæmia.

The effect of early removal of the teeth on the dental arches is shown in the three following cases :—

In the case shown in fig. 829 the deciduous molars were removed at the age of eight. The eruption of the premolars caused a slightly crowded condition, which was remedied by the extraction of the second premolars. There is here no evidence that a crowded arrangement of the teeth would have been avoided by the retention of the deciduous molars ; a line of treatment which produced a clean mouth for four years with, admittedly, some risk of crowding, which is not, however, difficult to remedy, seems strongly to recommend itself as against a septic mouth and possibly the avoidance of crowding.

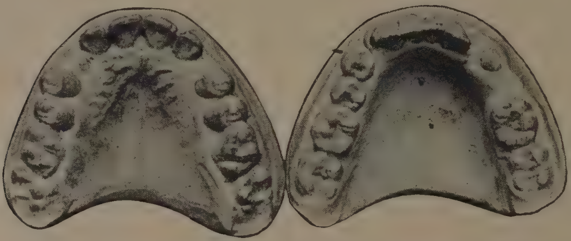

FIG. 829.—From *Royal Dental Hospital Reports.*

An example of a case in which the permanent first molars and the deciduous molars were removed at the age of nine years is shown in fig. 830.

Occasionally, extraction on these lines does not produce the desired results, but the failures are often to be traced to assymmetrical extraction rather than to the particular line of treatment.

Where the deciduous teeth are decaying on all surfaces—a condition not infrequently arising from over-indulgence in sweets, or the constant presence of fermentable carbohydrate on the surface of the teeth—a considerable improvement can be effected by careful regulation of the diet and regular cleansing of the teeth, combined with local treatment. For local treatment no drug is more efficacious than nitrate of silver, which should be applied to all the carious surfaces of the teeth after the removal of superficial caries. A small portion should be melted on the end of a silver probe so as to form a small bead which can be applied at any point with accuracy and safety. About four applications should be made at intervals of about a week, and afterwards at regular intervals of three months. A solution of sodium chloride should be applied to neutralise the silver nitrate remaining superficially on the teeth before the child is allowed to close its mouth, or the caustic effect of the silver salt may occur by contact on the tongue or cheeks. The use of carborundum stones on a dental engine to remove any sharp edges of carious teeth and render extensive cavities as self-cleansing and non-retentive as possible, is a great advantage when using silver nitrate for

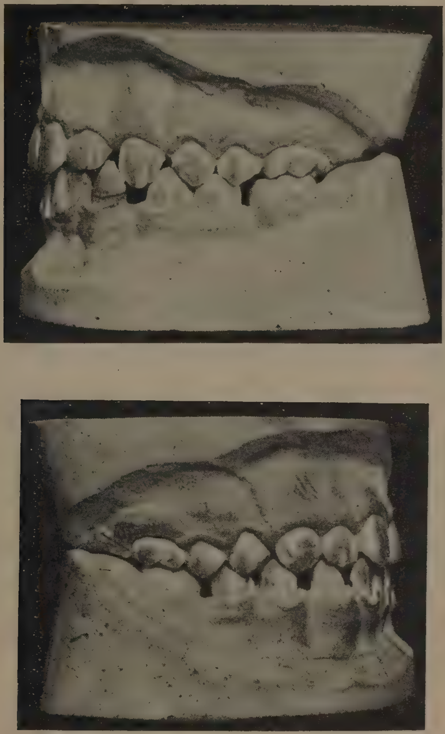

Fig. 830.—From *Royal Dental Hospital Reports*

extensive caries; the pulps of the deciduous teeth are not usually very sensitive, and the function of teeth so treated is usually rapidly restored in addition to the spontaneous arrest of caries being either aided or established, in which case the live pulps of these teeth, which otherwise would shortly have become exposed, efficiently protect themselves by the formation of secondary dentine. Twice a day, morning and evening, an alkaline mouth-wash should be used after brushing the teeth. If these directions are carried out, the teeth can often be retained for the normal period. On no account should nitrate of silver be held between the blades of conveying forceps, as it may slip from them and pass either into the larynx (as is known to have happened in one case), or into the stomach. If nitrate of silver passes into the larynx, inversion might be tried, but skilled surgical aid should immediately be sought; if it passes into the stomach, a plentiful supply of common salt should be given in order to create a chemical reaction leading to the formation of the insoluble and inert chloride of silver.

The practitioner should impress on parents the importance of keeping the permanent first molars scrupulously clean during eruption, and explain to them that these teeth form the vanguard of the second dentition, and that during their period of eruption the destructive agents are most active, the loose flap of gum overlying the crown acting as a food-trap.

If the permanent first molar shows signs of caries in the fissures on the occluding surface, the fissures should at once be cut out and filled with amalgam.

The removal of the deciduous teeth by extraction is dealt with in Chapter XXVII.

CHAPTER XXIV

Odontalgia and Neuralgia.

Pain arising in connection with disease of the teeth is usually designated by the term ‘ Odontalgia.’

Pain from visceral disease may be :—

(1) Local, *i.e.* located in the part diseased.

(2) Referred to the terminations of some nerve trunk implicated in the disease ; or

(3) ‘ Painful impressions may be conducted up the sensory fibres of the sympathetic into the central nervous system, and then referred to the peripheral distribution on the surface of the body of these nerve fibres that enter the same segment. This is true visceral reflected pain ’ (Head).

Pain from a tooth, therefore, may be localised to the tooth, referred to another part—for example, the temporal region—or associated with a painful area in the skin. It would seem, however, better to restrict the term ‘ odontalgia ’ to pain definitely referred to a tooth or teeth and to consider all cases in which the pain is referred to other parts as referred pain.

(A) ODONTALGIA.

Odontalgia may be divided into :—

(i) *Local.*

(ii) *Referred.*

By ‘ local odontalgia ’ is meant pain in or around teeth which are themselves the cause of the trouble ; by ‘ referred odontalgia ’ is connoted pain in a tooth which is not in itself the seat of the cause.

(I) LOCAL ODONTALGIA.

Nearly all morbid conditions of the teeth may be cited as causes of local odontalgia. For convenience they will be grouped under :—

(*a*) Morbid conditions of the periodontal membrane and parodontal tissues.

(*b*) Morbid conditions of the pulp.

The principal affections under the first head are acute and chronic periodontitis, parodontal disease and their sequelæ, and under the second, acute and chronic inflammation of the pulp.

Local odontalgia may be acute or chronic, and for practical purposes the source of the pain may be regarded as either the pulp or the periodontal membrane.

(1) Acute local odontalgia is generally due either to acute inflammation of the pulp or to acute periodontitis. *If due to inflammation of the pulp*, the pain will be of a sharp, shooting, throbbing character, more severe at night when the patient assumes the horizontal position and is falling off to sleep than at other times. Paroxysms of pain will also be caused by alternations of temperature. A small pledget of cotton-wool placed in the cavity and gently pressed upon with a blunt instrument will generally produce pain, as in this form of odontalgia the pulp is usually exposed. In a few cases the application of cold brings relief, while heat intensifies the pain. A condition of this character points to a suppurating inflammation of the pulp, the cold constricting the arteries and so reducing the local tension, the heat dilating the vessels and therefore increasing the local tension. A sharp, shooting pain, associated with marked tenderness of the tooth to pressure, usually implies tension extending from the pulp chamber via the apical foramina.

If periodontal or parodontal inflammation is the cause, the pain will be of a dull, gnawing, constant character. Percussion of the tooth with an instrument will generally cause pain. (Where pain arises in connection with the pulp, percussion does not as a rule cause pain.) Pressure with the finger upon the crown of the tooth will also produce pain, and the alveolar process is usually tender. A pledget of cotton-wool introduced into the cavity, if one exists, will not give rise to any pain except that which is caused by the pressure transmitted to the periodontal membrane. Alternation of temperature from hot and cold fluids may affect the pain, but not to the same extent as in inflammation of the pulp. By limiting the heat or cold to the tooth, the source of pain can be easily diagnosed. With periodontitis or parodontal disease pain will not be felt.

The *treatment of acute local odontalgia* depends upon the cause.

The remedy for each morbid condition has already been dealt with in previous chapters. Temporary relief of the pain in cases of pulp trouble may be obtained by applying to the cavity, on a piece of cotton-wool, some sedative, such as oil of cloves, oil of peppermint, or carbolic acid, and covering over the drug with some loose dressing, such as cotton-wool dipped in gum sandarac or calxine. Periodontal and parodontal pain can generally be relieved by the application of tincture of iodine, to which some tincture of aconite may be added, or by the local use of an adherent capsicum plaster. When the pain is due to tension in the pulp cavity from a putrescent pulp, relief can be obtained by opening the pulp chamber and so giving exit to its contents.

(2) Chronic Local Odontalgia.—The *causes*, like those of the acute form, may either be connected with the pulp or with the periodontal membrane. *The symptoms arising from the pulp* will be pain at irregular intervals, but less intense than in the acute form, and a tendency for the pain to wander and follow the course of the nerve.

Alternations of temperature, or the application of irritant food substances, such as sweets, sours, etc., generally produce a paroxysm of pain which may pass away at once or continue for some time. When the odontalgia arises *from the periodontal membrane or parodontal tissues*, the patient will complain of a grumbling sensation in the tooth and tenderness on pressure. The gum over the alveolar process will be slightly swollen, congested, and tender to pressure. The pain is usually constant, not paroxysmal, like the pain arising from an inflamed pulp, but it may be affected by alternations of temperature. A frequent cause of local odontalgia, and one that is often overlooked, is absorption of the septa between the teeth in parodontal disease. Food collects in the space formed, and pain results owing to pulp irritation via the cemental tissue or by infection via the periodontal membrane.

The *treatment*, as in the acute form, depends upon the cause.

(II) REFERRED ODONTALGIA.

Referred odontalgia may arise from many causes.

Any conditions which give rise to irritation of the terminal portions of the fifth nerve and its connections may cause reflex odontalgia, and by far the commonest condition which comes under this heading is that which is *dental in origin*—*i.e.* where the cause lies in a tooth which is not in itself the seat of pain. Such cases are of frequent occurrence. A patient complains of pain, say, in a mandibular molar, which, on examination, is found to be free from disease, the cause eventually being discovered in, perhaps, a maxillary tooth. This condition, generally called 'referred toothache,' is at times so pronounced that manipulation of the offending tooth will cause paroxysms of pain in the sound tooth, and in one case within recollection the application of arsenic to the pulp caused pain in the sound tooth during the process of devitalisation. Pain may be referred from maxillary to a mandibular tooth, or the reverse; it may also be referred from one tooth to another on the same side of the same jaw. Perhaps the most common and most instructive example of referred pain is that of a mandibular third molar causing symptoms in a mandibular premolar. Pain is never referred across the median line of the mouth. *Morbid conditions of the periosteum of the jaws, and ulcerations of the mucous membrane,* etc., may act as causes of odontalgia, and likewise *operations upon or morbid conditions of the eye and nose.* Cases supporting the latter statement are quoted by Galezowski and Macnaughton-Jones.

Toothache is frequently met with in patients with uterine troubles; in those with disorders of the alimentary tract, and in those who are the subjects of malaria, gout, rheumatism, and syphilis. The probable explanation is that the blood is charged with toxic matter, and slight lesions of the teeth cause pain which would not be felt under ordinary conditions; the altered nervous state may also be an important factor.

The *diagnosis of referred odontalgia* cannot always be readily arrived at. If no local cause can be found for pain which is referred by a patient to a tooth or teeth, a systematic examination of all the teeth must be carried out. As this examination is practically identical with the method to be pursued for investigating cases of neuralgia, it will be considered under that heading.

(B) NEURALGIA.[1]

Neuralgia is a term used to denote pain in the course of a nerve or within the area of its distribution. It is thus a somewhat vague term and does not connote any pathological condition. Since pain referred by the patient to the teeth is not necessarily the result of dental disease, and, on the other hand, pain originating in the teeth may be referred to other parts of the head, the diagnosis of facial neuralgia is clearly of great importance in dentistry.

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[1] For the revision of the description of the different clinical types of neuralgia the authors are indebted to W. Russell Brain.

Facial neuralgia may be classified as follows :—

1. Trigeminal neuralgia (Tic douloureux).
2. Neuralgia due to lesion of the trigeminal nerve and its branches.
3. Neuralgia due to lesions of the central connections of the trigeminal nerve within the brain stem.
4. Pain referred to the face and scalp from lesions of viscera including the teeth.
5. Facial neuralgia of psychological origin.

(1) Trigeminal Neuralgia.—Trigeminal neuralgia, or tic douloureux, is characterised by brief but intensely severe paroxysms of pain referred to one or more divisions of the trigeminal nerve. The cause of the disorder is obscure and no pathological changes have been identified in the trigeminal nerve. It most frequently attacks the middle-aged and elderly, and women are its victims more often than men. It is occasionally hereditary. It seems probable that dental sepsis may be a causative factor, but it is clearly not the only factor or trigeminal neuralgia would be much commoner than it is, and when once the disorder is established treatment of dental sepsis has no effect upon its course.

The paroxysms of pain begin in the second and third divisions of the trigeminal nerve with about equal frequency. In the course of time they tend to spread from one to the other and ultimately also to the first division. Characteristically they are brought on by talking, eating, a draught of cold air, and even washing the face. The pain frequently radiates into the upper or lower jaw and into the tongue. During the attacks, which last about half a minute, the face is often drawn up by a muscular spasm—hence the term 'tic douloureux.' The patient remains motionless, lacrimation is common, and salivation and sweating may occur. During the early stages remissions lasting weeks or months are not infrequent ; later the attacks occur in some cases many times a day.

According to Wilfred Harris,[1] it is commoner on the right side in the proportion of 3 to 2, and in females in the same or slightly higher ratio. When it occurs bilaterally the female is doubly prone to the disease.

Its incidence is very much higher in the United States than in Great Britain.

Dental treatment must be determined by the condition of the teeth, but, as already stated, it will not affect the course of the neuralgia. The only effective treatment for this is to interrupt the fibres of the trigeminal nerve either by injection of alcohol into the nerve trunks or Gasserian ganglion, or by surgical division of the sensory root.

(2) Facial Neuralgia due to Lesions of the Trigeminal Nerve and its Branches.—The trigeminal nerve is exposed to damage at various points. Both the supraorbital and the infraorbital nerves may be damaged in the course of fractures of the skull, and the infraorbital nerve is also exposed to infection from the maxillary antrum. Within the skull the Gasserian ganglion may be the site of inflammation in herpes zoster, which, during the acute stage, causes a vesicular eruption over the face and may be followed by intractable neuralgia. Within the skull also the nerve may be compressed by a tumour or involved in meningeal inflammation in syphilis.

Neuralgia due to organic disease of the trigeminal nerve is usually characterised by a more or less continuous background of pain with more severe paroxysms which last much longer than those of tic douloureux. Moreover there is likely to be some change in the sensibility of the skin, either in the direction of hypersensitiveness or relative anæsthesia. Damage to the motor root of the trigeminal nerve will cause wasting of the muscles of mastication and deviation of the jaw to the affected side on opening the mouth.

(3) Facial Neuralgia due to Lesions of the Central Connections of the Trigeminal Nerve within the Brain Stem.—Very severe pain may be referred to the face as a result of lesions which irritate the pain fibres derived from the trigeminal nerve after they have entered the brain stem. Fortunately such conditions are rare, but for this reason they are apt to be wrongly diagnosed. Syringobulbia, tumours of the medulla, vascular lesions secondary to arteriosclerosis and disseminated sclerosis are the principal causes. In such cases again there will usually be an impairment of sensibility, especially to pain, heat and cold, over some part of the skin of the face.

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[1] *Brit. Med. Journ.*, 1935, ii, 592.

(4) Pain referred to the Face.—Numerous workers, especially Head, have demonstrated that disease of a viscus may give rise to pain which is located on the surface of the body at a point remote from the diseased viscus, and may be associated with hypersensitiveness of the skin of this area to the prick or scratch of a pin. This phenomenon is known as 'referred pain' and seems likely to be illuminated by the recent work of Lewis on what he has termed the 'Nocifensor System' of nerves. Lewis has demonstrated that a lesion of the skin often evokes by means of an axon reflex the liberation into surrounding areas of skin of a substance which is capable of stimulating the ends of the pain fibres.

Head showed that lesions of the teeth were capable of causing pain which was referred to regions of the face and scalp, and that to some extent there was a correlation between individual teeth and the sites into which pain from them was referred.

The exact relation of each tooth to the segmental areas seems to vary. The following table is given as approximate :—

MAXILLA.	
Incisors	Fronto-nasal.
Canine	Naso-labial.
Premolar I.	,,
,, II.	Temporal or maxillary.
Molar I.	Maxillary.
,, II.	Mandibular.
,, III.	,,

MANDIBULAR.	
Incisors	Mental.
Canine	,,
Premolar I.	,,
,, II.	Doubtful.
Molar I.	Hyoid.
,, II.	,,
,, III.	Superior laryngeal or hyoid.

The diagrams (fig. 831) show these areas, and the maximal points of these areas.

An unerupted mandibular third molar may cause pain which is referred not only to the side of the neck, but over the shoulder and greater part of the upper limb.

As Head showed, not only the teeth, but the nasal sinuses, the eye, the tongue and the tonsil may also cause pain which is referred to the head, and the same is true of remote viscera, such as the heart and lungs, and even those within the abdominal cavity. Disease of the apex of the lung may cause severe neuralgic pain which is referred to the mandible on the same side and has in some cases led to extraction of teeth because the true origin of the pain was not recognised.

(5) Facial Pain of Psychological Origin.—There is an important group of patients, mostly female, who complain of agonising pain referred to the face or jaw for which no organic basis can be found. Not uncommonly the start of such a pain is said to have been extraction of a tooth. Sometimes further teeth have been extracted in the effort to abolish it, without success. The patient complains that no analgesic drug will relieve the pain, and her emotional attitude to it will often provide a useful clue as to its nature. Pain in these cases is a

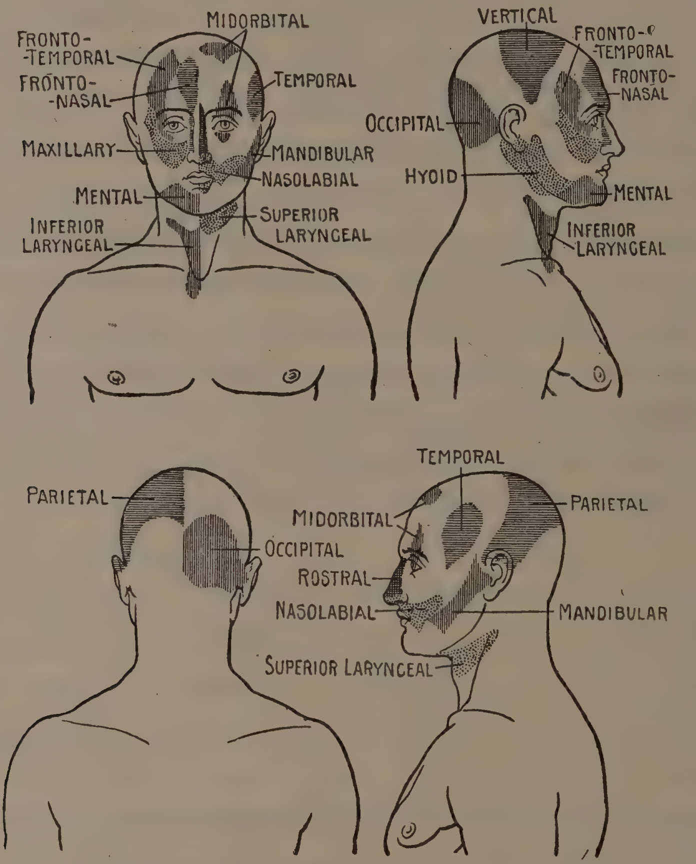

Fig. 831. (From Allbutt's 'System of Medicine.')

hysterical symptom and can only be satisfactorily treated by psychological methods.

Diagnosis.—The diagnosis of the cause of facial neuralgia is important, because the treatment must depend on the cause, and all cases which come under notice should be subjected to a systematic examination. The patient should be questioned as to the character of the pain, in order to discover whether the pain arises from the pulp or from the periosteum. It is also important to ascertain if the pain is periodic, while at the same time the knowledge of its situation is useful in assisting to localise the position of the tooth should the neuralgia be of dental origin. Thus, pain in the infraorbital region points to maxillary teeth; pain in the ear, shooting down the shoulder, points to mandibular teeth; while pain over the parietal eminence indicates trouble from either maxillary or mandibular teeth. Next, the teeth must be examined for carious cavities, and, should any be found, a search should be instituted to discover if any chronically inflamed pulps exist. This examination should be carried out with a mirror and probe, care being taken to look for approximal cavities near the gum margin. After this, parodontal causes should be excluded by pressing and tapping the teeth and carefully examining the 'pockets' around the teeth. Irritability of the pulp through pathological changes must next be eliminated by looking for exposed surfaces of dentine or cementum. Exposure of the root in local parodontal disease is a fruitful cause of neuralgia (see p. 482). Each tooth should next be tested with heat and cold, and, in doing this, attention should be bestowed upon teeth containing large metal fillings. In testing with heat, a ball of base-plate gutta-percha should be made hot and applied to each tooth separately; while the test with cold is easily carried out by touching each tooth with a small pledget of cotton-wool dipped in ice-cold water, or spraying with ethyl chloride. If a jet of water is used from a syringe, the water will run over two or three teeth at a time, and so prevent ready detection of the offending tooth. It must not be overlooked that most teeth with live pulps, if submitted to the above treatment, will respond to the changes; but when the pulps are healthy, the condition will be quite transient, while, if diseased, the test will probably set up an acute paroxysm of pain. In cases of doubt the neighbouring teeth should be tested as well as the suspected tooth. The pain started in the affected tooth will be greater in comparison with that in the approximal teeth. Suppurative conditions of the periodontal membrane are to be regarded as frequent causes of trigeminal pain. The exposure of the roots leads to irritability of the pulps from thermal and other irritations, while in advanced conditions the pulps may be affected via the apical foramina by spread of inflammation from the periodontal membrane.

If a cause has not been discovered by the above-mentioned examination, the presence of unerupted third molar teeth, a frequent cause of neuralgia in those between eighteen and thirty, should be considered. Such causes as morbid conditions of the periosteum of the bony canals through which the nerve passes, or peripheral irritation in other parts of the distribution of the fifth nerve, must next be excluded. The presence of disease of the maxillary sinus or tumours in the region of the trunk of the nerve should be looked for, and the question of organic disease in the brain and trigeminal neuralgia must be considered.

Radiograms are an invaluable aid in such an examination; unseen caries, buried, misplaced, or supernumerary teeth, apical rarefactions, neoplasms, cysts, and other morbid conditions of the jaws or teeth which might not otherwise be suspected, may be disclosed by radiogram.

A case which occurred in the practice of the late F. E. Sprawson is illustrative. A nurse aged about thirty-six had been treated by many physicians and dental surgeons for a violent and intractable maxillary neuralgia over a period of more than ten years; the effects were that in spite of her age she appeared a white-haired and old woman, and was willing to undergo any form of treatment to obtain relief; a radiogram disclosed an unerupted maxillary premolar which had impinged on and caused absorption of its neighbouring premolar till it had reached the pulp; on the removal of both premolars the neuralgia ceased immediately.

Treatment.—If the neuralgia is dental in origin, the cause must be removed by the measures indicated for the treatment of such lesions. When the cause is constitutional, remedies appropriate to the condition must be adopted. For instance, if arising in connection with general debility, anæmia, or overwork, a general tonic treatment with such drugs as iron, quinine, and arsenic will be beneficial, careful attention being paid to the condition of the bowels. Aspirin (10 to 15 gr.) is useful in neuralgias of peripheral origin. If of malarial origin, quinine in large doses, *i.e.* 5 to 10 gr., will bring immediate relief, and, if given before an attack, will, in some cases, ward it off, and, in others, greatly minimise its severity. Should there be any suspicion of syphilis, iodide of potassium would be indicated; while with gout and rheumatism the same drug may prove useful. In addition to the internal administration of drugs, local applications may be tried, such as chloral and camphor in equal parts, aconite and chloroform, or counter-irritants, such as cantharides, capsicum, and mustard. If no cause can be found, and the above treatments fail, the drugs which may be termed ‘ neuralgic specifics ’ should be tried, amongst these being tinct. gelsemii, veratrina, butyl chloral hydras. In combination with these, such drugs as dilute hydrobromic acid, chloral, and bromide of potassium may be given. In more severe cases, the injection of morphia may be resorted to.

In cases where treatment by drugs fails and the patient continues in great pain, injections of alcohol may be tried, and failing relief from these, surgical methods may be adopted. These questions belong to the domain of general rather than dental surgery.

CHAPTER XXV

The Remote Effects of Disease about the Teeth.

By MARTIN A. RUSHTON, M.B., B.Ch. (Cantab.), L.D.S. (Eng.).

Focal Infection—Paths of Influence—Open Sepsis—Material Swallowed, Inhaled—Closed Sepsis, Acute and Chronic Infection—Remote Effects—Cardiovascular System—Rheumatic Diseases—Central Nervous System—The Eye—The Skin—Principles of Treatment.

(A) FOCAL INFECTION.

INFECTION of almost any part is a concern of the body as a whole. In the case of acute local infections the relationship is very apparent: a septic finger, a carbuncle, or an acute alveolar abscess gives rise to a feeling of illness, to fever, headache, loss of appetite, and to characteristic changes in other tissues. In the case of chronic local infections the remote effects are much less obvious, but there is ample evidence that the absorption from them of organisms or their toxic products may produce serious damage in distant tissues. Such areas of chronic local disease are called in this connection foci of infection or of sepsis, and may be situated in such parts as the cervix uteri, the antrum, or the tonsils and the supporting structures of the teeth.

Though infection about the teeth may often appear to produce no damage to the body as a whole, it is only the most trivial local infection which does not place a strain on the defensive mechanisms of the body for its repair or arrest. So long as these mechanisms are intact and the infection is not too severe the remote effects of oral sepsis are not apparent; but the potential danger of even a minor lesion becomes evident in those cases where intercurrent disease or age has weakened the powers of resistance. Almost any infection about the teeth, then, may in certain circumstances lead to remote effects, but the results produced will depend upon the nature of the initial infection, the path by which the septic products are distributed, the constitution of the patient, and such factors as superadded disease and injury.

(B) THE PATHS OF INFLUENCE.

The spread of infection by continuity of tissue, leading, for example, to inflammation of the antrum, Ludwig's angina, osteomyelitis of the jaws and the like, and the reflex results of irritation are described elsewhere ; and specific infections which may have their primary focus in the mouth, such as diphtheria or syphilis, will not be mentioned here.

Open Sepsis is a term used to denote an infected or septic condition in direct communication with the buccal cavity, where the toxic products drain into it. This category includes all superficial lesions, the sepsis resulting from the accumulation of calculus, and the poisonous amines of food decomposition, and deeper lesions communicating freely with the mouth. Such infection may influence other parts by being swallowed, by inhalation, or by extending along the surface to the nasopharynx and ear or up the ducts of the salivary glands.

Closed Sepsis refers to those lesions which are not in direct communication with the oral cavity, and where extension or absorption may be expected to occur through the blood and lymph vessels. In this group infection of the alveolar bone occupies an important place.

The distinction between ' open ' and ' closed ' sepsis is one of convenience and is not rigid. Thus in parodontal disease there is both drainage into the mouth and absorption into the lymphatics and veins.

(C) OPEN SEPSIS.

The Effects of Swallowing Septic Material.

Pathogenic bacteria from a septic mouth may continually reinfect the **tonsils** or contaminate the operation wound of tonsillectomy ; and the continual passage of septic material promotes **pharyngitis.**

It has been established that in the normal subject micro-organisms swallowed are rapidly destroyed by the acid gastric juice (except perhaps at night) and do not survive in the stomach or pass into the small intestine. The fate of the toxic products of decomposition which accompany them is more problematical. When the swallowing of septic material continues for a long time, however, the stomach may become damaged. The changes which occur are apparently due to prolonged irritation rather than to any specific infection, and may also be produced by inefficient mastication of food through lack of teeth, faulty artificial dentures, or hurried meals; the use of much highly spiced, very hot or cold foods; excessive addiction to tobacco and alcohol and the like.[1] The response to these insults depends upon the inborn constitution of the individual. In those of the so-called *hypersthenic* gastric constitution the result is an increased secretion of hydrochloric acid (**hyperchlorhydria**), sometimes leading in conjunction with psychological and other factors to **gastric ulcer,** which in a certain number of persons undergoes malignant degeneration. It is believed that about 30 per cent. of cases of **gastric carcinoma** arise in this way. When gastric ulcer is already present, untreated oral infection retards or prevents its healing.

In patients of *hyposthenic* type the course of events is completely different, and the results are chiefly due to the impairment of one or more of the many functions of the gastric mucosa. **Chronic gastritis** occurs, accompanied first by the loss of acid secretion **(achlorhydria)** and then by the absence of pepsin. This not only causes indigestion but permits the passage of bacteria from the mouth into the duodenum, which is usually sterile, and the rest of the gut. Increased alkalinity of the small intestine favours an ascending growth of *Bacillus coli*, so that infection of the **gall bladder** and other parts with either this organism or a streptococcus from the mouth is now possible.

Chronic achlorhydric gastritis is the common forerunner of **gastric carcinoma** when this does not arise on the site of a simple ulcer, that is in the remaining 70 per cent. of cases. Steadman[2] long ago observed the high degree of dental sepsis found in sufferers from carcinoma ventriculi. Herbert and Bruske[3] more recently compared the oral condition and habits of 500 patients at Guy's Hospital with a similar number at the Wilhelmina Gasthuis, Amsterdam, and correlated the much higher incidence of gastric carcinoma in Holland with a much greater degree of oral sepsis and masticatory inefficiency, and a more generous employment of spirits, tobacco, and very hot and spiced foods.

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[1] A. Hurst, *Brit. Med. Journ.*, 1937, ii, 783.

[2] F. St. J. Steadman, *Brit. Dent. Journ.*, 1923, December 1.

[3] W. E. Herbert and J. J. Bruske, *Guy's Hosp. Reports*, 1936, lxxxvi, 301.

Another sequel of gastric achlorhydria is a deficient absorption of iron from the food leading to **simple achlorhydric (microcytic) anæmia.**

In some persons the swallowing of septic material and other irritants leads to impairment of further functions of the stomach, such as the production of factors which normally prevent **Addison's (pernicious) anæmia** and **subacute combined degeneration of the spinal cord.**

In all cases where operation on the alimentary tract is contemplated, preliminary attention to the health of the mouth is of the greatest importance, since the gastric secretion will be inhibited for a day or two after the operation and infection of a gastric or intestinal wound may well occur. The dental treatment should be no half-measure and should include the elimination of both ' open ' and ' closed ' sepsis, since post-operative infection is likely to be disastrous.

The Inhalation of Infected Material.

The inhalation of infected particles or fluids from the mouth is one of the dangers of general anæsthesia, and may result in a **pulmonary abscess** or **septic broncho-pneumonia,** either of which may be fatal. For this among other reasons the mouth should receive attention before such an anæsthetic is given in all except cases of great emergency, and where the site of operation is to be within the mouth the oropharynx should be adequately packed.

The entry of mouth organisms such as streptococci into the respiratory passages is also possible in conscious patients, and in those of poor resistance may lead to broncho-pneumonia, **bronchitis,** or laryngitis, while the condition of those already afflicted with tuberculous pulmonary lesions or bronchiectasis will be made worse by the additional mixed infection. The presence in the lungs, usually in gangrenous areas, of *B. fusiformis* and a spirochæte has often been described, sometimes in patients with Vincent's infection of the mouth. The relation of oral sepsis to **asthma** is difficult to assess owing to the important part played by psychological factors in many cases, but sensitisation to mouth organisms sometimes appears to be responsible. **Puerperal fever,** usually a septicæmia due to a hæmolytic streptococcus, is commonly caused by droplet infection from the mouths and throats of those attending cases of childbirth, and occasionally by direct transference from the patient's own infected saliva. A blood-borne infection of the pelvic organs from a dental focus may occur in rare cases. A healthy mouth is essential for all engaged in the practice of midwifery.

Extension by Other Passages.

Except when due to specific infections such as mumps, **sialoadenitis, parotitis** commonly and infection of the other salivary glands occasionally, is nearly always due to the ascent of the ducts by bacteria from the mouth. An important predisposing cause is the arrest of salivation which may follow surgical operations on the alimentary tract and starvation, or accompany fever. Care of the mouth before operations and in conditions such as œsophageal obstruction where oral feeding has to be abandoned, together with salivary stimulation, has greatly reduced the incidence of acute parotitis, which was formerly considerable. Dirty or ill-devised artificial dentures abutting on the orifices of salivary ducts, accumulations of calculus in the same regions, and a septic state of the mouth, are important causes. The dental surgeon sometimes sees cases of acute parotitis following fractures of the jaw or neglected infection about a third molar where the patient has been unable to chew or clean the mouth adequately. In acute cases the organism is usually *Staphylococcus aureus*, and in the recurrent *Streptococcus viridans*.

Infection may spread to the nasopharynx and thence along the Eustachian tube to the middle ear, producing **Otitis media,** the prolonged suppuration of which will sometimes respond to no treatment until the mouth has been rendered healthy.

(D) CLOSED SEPSIS.

Acute Infection.—The general disturbance produced by acute infection about the teeth resembles that occurring in conjunction with acute infection elsewhere. There may be severe intoxication without manifest dissemination of bacteria, causing, for example, fever or delirium. But if the local infection be not overcome the micro-organisms may spread along the lymphatics, producing **acute lymphadenitis,** which may become suppurative ; or by the veins, leading to **septicæmia,** to septic **thrombosis of the cavernous sinus,** to **acute bacterial endocarditis,** to **pyæmic abscesses** of the lungs or other secondary focus. Although acute infection may spread in this way unprovoked in persons of poor resistance, such extension can be precipitated by ill-judged or unfortunate interference, which may suddenly introduce infection into new areas where no resistance has been prepared. Thus the extraction of teeth in the presence of acute stomatitis or the removal of much adjacent bone in the early stage of abscess formation may have this effect. Conversely, the neglect to provide early drainage for an acute abscess may lead to the same result. According to Rousseau-Decelle, an important cause of acute septicæmia is the reckless filling of teeth of which the pulps have already become infected, especially in the lower jaw. Cases following incautious instrumentation in root-canals are also on record.

In acute septicæmia the extension of infection is announced by severe rigors, a sudden rise of temperature, and a rapid pulse of low tension. Dyspnœa and delirium soon appear, followed by collapse and death in a few days. The organism concerned is usually a streptococcus, frequently hæmolytic, but sometimes a staphylococcus as in the following case :—

A schoolboy had ' toothache ' in a mandibular premolar which had been rather heavily restored, and the tooth was extracted under nitrous oxide anæsthesia. Nothing untoward happened for three days and then swelling appeared which spread along the jaw from the symphysis to the articulation on the same side. An incision was made but no pus was evacuated. The remaining teeth on that side of the mandible became loose and the periosteum was stripped up. The mouth was intensely dry and hot, and the boy had a high, swinging pyrexia. A drainage-tube was inserted from the original incision right down to the jaw, but no pus was found. Blood-culture gave a pure growth of *Staphylococcus aureus* ; and from the apices of the teeth, which had become so loose that they could be picked out with dressing forceps, a heavy growth of *S. aureus* as well as the usual *Streptococcus viridans* was obtained. The boy died.[1]

In the less fulminating cases secondary localisation of staphylococcus, streptococcus, or pneumococcus may occur as in this case :—

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[1] A. Bulleid, *Lancet*, 1936, i, 931.

A boy aged 10 years suffering from congenital pulmonary stenosis was sent to Guy's Hospital by his physician for dental treatment. For 3 weeks while under observation in the ward his rectal temperature remained below 100°. On Dec. 2 one tooth was extracted, and on the 3rd the temperature rose to 101°. On the 4th two more teeth were removed and the temperature rose to 102° and 103° on the following days and remained at a high level. On the 7th hemiplegia occurred and on the 14th he died. Autopsy showed evidence of acute bacterial endocarditis and two cerebral emboli.[1]

Here the congenital abnormality of the heart determined the site of secondary localisation. Thrombosis of the cavernous sinus arises from a more local venous spread either along the facial and angular veins or through the pterygoid plexus and foramen ovale, but signs of general septicæmia are usually present in addition. Death occurs in from one to three weeks.

Acute hæmorrhagic nephritis may result from an acute alveolar abscess in which a hæmolytic streptococcus is concerned, and **rheumatic carditis** may be observed in the same circumstances.

Some cases strongly suggest that the jaws may occasionally provide the primary focus for the common staphylococcal **osteomyelitis** of the long bones of children. Bulleid [2] cites such a case :—

A boy of 14 received a blow on his right shin during a game of hockey. The leg was painful for a few days and showed some bruising, but there was no fracture. The bruising and swelling soon disappeared and no further notice was taken of the injury. Some six weeks later the boy developed an acute alveolar abscess in connexion with a maxillary premolar tooth, which was extracted. Cultures from the evacuated pus gave a heavy growth of *Staphylococcus aureus* associated with *Streptococcus viridans*. Recovery from the extraction was uneventful, but about a week later the right leg became painful at the site of the original blow. A surgeon was called in and diagnosed osteomyelitis, which was confirmed by operation. It was suggested that the staphylococcus gained access to the blood at the time of the extraction of the tooth and that the previous injury to the leg localised the organism.

Fish [3] takes the view that secondary localisation of this kind only occurs if the organism is actually circulating in the blood at the time and place that the injury is received. In the above case, for example, he suggests that a further trivial injury of the original bruise immediately after the extraction would permit localisation.

[1] M. A. Rushton, *Guy's Hospital Reports*, 1930, lxxx, 39.
[2] A. Bulleid, *loc. cit.*
[3] E. W. Fish, *Proc. Roy. Soc. Med.*, 1937, xxx, 1149.

Chronic Infection.

While the manner in which acute dental disease affects other parts of the body is relatively clear, the mode of action of chronic infection remains more obscure.

In a few cases organisms of dental origin are carried to distant tissues and there proliferate, forming secondary foci. Such is the case, for example, in subacute bacterial endocarditis, where the *Streptococcus viridans* sets up a secondary focus on damaged heart valves.

In other cases it seems more likely that specific harmful effects are rather due to the absorption of toxic products from the primary focus. Urticaria and, in the opinion of many, rheumatoid arthritis arise in this way.

In the third and largest group the effects of oral sepsis are not specific, but so injure or occupy the defensive mechanisms of the body that its power of resisting other disease, or the changes associated with age and degeneration, is impaired. Thus it is found more difficult successfully to treat nearly every disease when infected teeth are present, though it is not suggested that this infection is the cause of the general disease ; indeed, it may sometimes be the result of it.

Foci of Chronic Infection.

The sources of infection and intoxication in ‘ open sepsis ’ are apparent, but those in ‘ closed sepsis ’ require further description.

Pulpless Teeth.

The apices of untreated pulpless teeth are always infected, whether a chronic abscess be present, walled off by fibrous tissue, a granuloma, a dental cyst, or a diffuse zone of osteitis. Absorption of toxic substances into the body fluids can occur, and the spread of organisms may also be possible. The condition of the apices of treated pulpless teeth is often similar to that of the untreated, and radiograms may show evidence of osteoporosis or sclerosis, usually but not always the result of infection. The state of those apices which appear relatively normal in a radiogram, however, is a matter of dispute. While some contend that a treated pulpless tooth which shows none of the radiographic appearances associated with inflammation is not to be regarded as a possible source of infection or intoxication, others consider that radiographic criteria are of little value in this connection. Bulleid,[1] as the result of examining a large number of such teeth bacteriologically, both extracted and *in situ*, formed the opinion that the absence of evidence of apical osteitis in a radiogram does not indicate the absence of infection, and that no conservative procedures yet known are capable of preserving sterility in pulpless teeth for any length of time, except possibly apicectomy in the case of single-rooted maxillary teeth.

What has been said of pulpless teeth applies also to portions of teeth left embedded in the jaws.

Residual Infection.

It may happen that after diseased teeth have been removed infection of the alveolus still persists, due, perhaps, to preceding necrosis or to deficient powers of recovery. Such infected tissue, which may show a characteristic ‘feathery’ appearance in radiograms and is often associated with delay in the rearrangement of bone that normally follows the extraction of teeth, may be a source of further intoxication and infection. It sometimes proceeds to a frank chronic osteomyelitis of the jaw. It is by no means certain, however, that the radiographic appearance mentioned always indicates present infection of the bone.

Parodontal Infection.

There is no doubt that the infection present in parodontal disease is a source of danger to the body as a whole. Apart from the harm which may follow the swallowing or inhalation of the bacteria concerned there is evidence that they are also discharged freely into the blood stream. Okell and Elliott [2] on culturing the blood of patients with severe parodontal disease obtained growths of *Streptococcus viridans* in 10 per cent. of cases. And Round, Kirkpatrick, and Hails [3] found, on examining the blood of two similar patients before and after hard sweets had been chewed, that bacteriæmia could be induced by mastication.

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[1] A. Bulleid, *loc. cit.*

[2] C. C. Okell and S. D. Elliott, *Lancet*, 1935, ii, 869.

[3] H. Round, H. J. R. Kirkpatrick and C. G. Hails, *Proc. Roy. Soc. Med.*, 1936, xxix, 1552.

While the fact of this bacteriæmia, which may be transitory or intermittent, is not in dispute, there is some disagreement as to the precise source of the organisms. It has commonly been believed that the rarefied alveolar bone surrounding the teeth is itself infected, and is to a great extent the source of septic absorption. But some have strongly denied this, and recently Fish [1] has put forward the view on histological and experimental grounds that this bone is not the seat of sustained infection, that the source of the bacteriæmia lies in the necrotic surface cells and debris of the pocket, and that bacteria are thence expressed by the force of mastication or other cause into the general circulation. The absorption of toxic products occurs in the absence of such trauma, and the rarefaction of the alveolar bone he believes to be due to this cause. The settlement of this point is of great importance in deciding to what extent conservative treatment of parodontal disease is justified in cases where remote effects are likely.

Infection of the Apices and Pulps of Vital Teeth.

Dowsett,[2] Bulleid,[3] and others have recorded the infection and absorption of the apices of teeth with vital pulps. In four out of six cases a hæmolytic streptococcus was cultivated after open operation or extraction. The roots of many teeth were affected progressively, and treatment for phlebitis or arthritis was not successful until the teeth had been removed and an autogenous vaccine given.

There is some doubt as to whether vital teeth showing cementosis should usually be regarded as infected. Gardner and Goldstein,[4] after examining many hundreds of such teeth, expressed the opinion that the hyperplasia was rather due to an inherited tendency in certain persons than necessarily a response to infection. A familial distribution of the abnormality has been observed.

The question whether a vital pulp may act as a focus of infection has been disputed. Bulleid and Ellingham [5] were able to cultivate bacteria from the pulps of 22 out of 30 heavily restored vital teeth, but the presence of organisms in the pulps of extracted teeth does not necessarily indicate infection unless histological evidence points in the same direction. Tunnicliffe and Hammond [1] isolated streptococci from the pulps of 10 out of 30 intact teeth, but the pulps showed no leucocytic infiltration. It is likely that in such cases infected lymph may have been forced into the pulp during the extraction of the tooth.

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[1] E. W. Fish, *loc. cit.*

[2] E. B. Dowsett, *Proc. Roy Soc. Med.* (Odonto. Sec.), 1924, xvii, 6.

[3] A. Bulleid, *loc. cit.*

[4] Boyd Gardner and H. Goldstein, *Dental Cosmos*, 1931, lxxiii, 1065.

[5] G. Ellingham, *Brit. Dent. Journ.*, 1936, lx.

Unerupted Misplaced Teeth.—The follicles of unerupted misplaced teeth may become centres of infection when they are near to the surface and occasionally when they are more deeply situated. There is no ground for assuming that they are always infected.

The Bacteriology of 'Closed Sepsis.'

The organisms concerned are believed to be principally the streptococci, the staphylococci being occasionally involved. Bulleid [2] states that no other organisms which can be cultivated from dental lesions give rise to specific antibodies in the blood-serum of the patients concerned.

In the acute apical infections a higher proportion of true β-hæmolytic streptococci is found than in the chronic lesions, where *Streptococcus viridans* and indifferent types prevail. Bulleid invariably found the latter varieties in cases of residual infection. In those dental lesions in which *Staphylococcus aureus* was demonstrated in symbiosis with a streptococcus it was noticed that severe constitutional disturbance followed the removal of teeth thus infected.

(E) REMOTE EFFECTS OF CHRONIC FOCI.

The number of diseases which have been ascribed to a blood-borne dissemination of the bacteria or toxic products of oral infection is enormous. While improvement in almost every disease follows the careful treatment of concurrent infection about the teeth, this is usually due to the removal of an additional burden which the body has had to bear rather than to elimination of the cause of the disease. Nevertheless there is reason to believe that in some maladies there is a more direct relationship.

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[1] R. Tunnicliffe and C. Hammond, *Journ. Amer. Dent. Assoc.*, 1937, xxiv, 1663.

[2] A. Bulleid, *loc. cit.*

Evidence of such relationship in any particular case usually rests on clinical observation. In a few cases it may be possible to isolate the same micro-organism from the oral and remote lesions. The results of agglutination tests using streptococci isolated from oral foci and the same patient's serum, though sometimes positive in a high dilution, cannot yet be interpreted with certainty; but a rise in the sedimentation rate of red corpuscles is an indication that a focus has more than local importance. An exacerbation of the remote symptoms following operation on a suspected oral focus or the injection of a vaccine prepared from such a site is highly suggestive of a relationship, and is sometimes clinically so important that the greatest care must be exercised in order that operations may not do more harm than good. The disappearance of remote symptoms after the successful treatment of oral infection, though very satisfactory to all parties, is not by itself an indication that there was any direct connection between the two. And, conversely, the persistence of symptoms in spite of dental treatment does not mean that oral disease was not responsible for their first appearance, since a secondary focus may have been set up, a vicious circle initiated, or irreparable damage already done.

Rosenow and his school have put forward a concept which they call the elective localisation of bacteria. By this they mean that micro-organisms which have attacked a certain organ, such as a joint, will, if newly isolated and injected into an experimental animal, attack the same tissue in the new host. Further, they claim to have shown that bacteria obtained from the teeth of patients who suffer from various remote lesions such as arthritis and cholecystitis will produce similar lesions in experimental animals. These experiments have been offered as proof that such lesions are in fact the result of infection about the teeth in the cases investigated.

(F) CHRONIC INTOXICATION.

Probably a common result of chronic oral disease is a general impairment of vitality without recognisable peripheral lesions. 'Probably,' because there is rarely clear evidence that this is the case, though it appears very likely. Of patients sometimes classified as 'pyrexia of unknown origin' a number are suffering from intermittent streptococcal bacteriæmia and continual intoxication from a dental source, commonly advanced parodontal disease. They are always tired, from time to time have slight evening fever, night-sweats, digestive disturbances, or albuminuria. The symptoms are those of septic absorption from any source. The patients become thin and sallow and steadily deteriorate until some organ is no longer able to bear the strain, or they are attacked by an additional infection; but if they receive suitable dental treatment they rapidly improve.

In children chronic disease about the deciduous teeth causes loss of appetite, slight fever, and failure to maintain a normal increase in weight.

Chronic Lymphadenitis, as well as the acute form, is often caused by infection about the teeth, it being the function of the lymphatic glands to destroy toxic material carried to them by the lymph vessels. Often the latter are also chronically inflamed and may even suppurate.[1] The glands affected in the first instance are of the submental or three submaxillary groups, according to the position of the oral lesion, but other groups may become inflamed in succession or occasionally. An association between dental disease and tuberculous lymphadenitis has long been observed, and it has been reasonably suggested that the presence of unhealthy teeth and gums makes tuberculous infection more likely.

(G) CARDIOVASCULAR SYSTEM.

Subacute Bacterial Endocarditis.—Where the endocardium has been damaged by acute rheumatism, or in the presence of a congenital cardiac abnormality, secondary infection may occur at the margin of the valves. By far the commonest infecting organism is *Streptococcus viridans*. Many cases have been reported in which the disease commenced soon after the extraction of a number of septic teeth, and the work of Elliott [2] supports the belief long held that these operations were the precipitating cause. He found that a positive blood culture of streptococci could be obtained in 75 per cent. of cases of severe parodontal disease for about ten minutes after the extraction of teeth. If the large number of organisms liberated into the circulation by such an operation in an infected mouth may be sufficient to set up a secondary focus on the damaged endocardium, the lesser bacteriæmia associated with dental foci when no operation has been performed may have the same effect. It is believed that secondary localisation does not take place unless there is a freshly formed thrombus on the valve. The condition is fatal, but may run a course of several years. The patient becomes anæmic and feeble with intermittent fever, and by reason of the infected emboli which are discharged from the valves sustains infarcts in all parts of the body, kidneys, brain, spleen, lungs, skin, etc. The following is a report of a post-operative case in which symptoms of bacterial endocarditis arose relatively early :—

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[1] E. W. Fish, *Brit. Dent. Journ.*, 1935, lviii, 531.

[2] C. C. Okell and S. D. Elliott, *loc. cit.*

A boy 4 years old was admitted to Guy's Hospital in June 1923 suffering from acute rheumatism with pericarditis. He recovered and was discharged in October. He had at that time mitral stenosis and regurgitation and his teeth were in good condition. He was readmitted in August 1925 for the extraction of four septic teeth, was then looking and feeling well and had the same cardiac signs as before. On August 22 the teeth were removed. For three days the temperature remained normal, then rose to 102·6° and persisted at a high level. On September 15 blood culture showed abundant colonies of *Streptococcus viridans.* Thereafter signs of subacute bacterial endocarditis including Osler's nodes appeared and in January hemiplegia occurred. He died in March.[1]

In patients with cardiac lesions it is thus of the greatest importance that caries shall not be allowed to reach the pulp and that the gums shall be kept healthy. The devitalisation of teeth should not be undertaken ; and when extraction is necessary the mouth should first be cleaned, only a few teeth should be removed at once and with as few rocking movements as possible, and any pockets around these teeth should be disinfected before the operation. Fish [2] strongly recommends that the actual cautery be used for this purpose. Medical co-operation with regard to sulphonamide or other chemotherapy should also be invited. When signs of bacterial endocarditis have already appeared the extraction of teeth is generally both useless and unkind. Cases have been reported in which functional irregularity of the heart has been ascribed to infection about the teeth. In so far as such foci contribute to general ill-health they may be an accessory cause, but there is very little evidence of a direct relationship to myocardial damage except in the case of pyæmia. Heart failure may follow the removal of a large number of septic teeth from weak patients as it may any other severe operation.

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[1] M. A. Rushton, *loc. cit.*

[2] E. W. Fish, *Proc. Roy. Soc. Med.*, 1937, xxx, 1149.

Foci of sepsis are also among the many contributory causes of angina pectoris and **arterial hypertrophy** associated with raised blood pressure.

The source of infection in **phlebitis** may be in the jaws.

A patient developed phlebitis in his right leg, which in time cleared up. The only history he gave was that he had previously grazed his heel on a nail in his boot, and this soon healed. Later the phlebitis recurred and yet no obvious focus of infection was found. His teeth were radiographed and it was discovered that one of the mandibular premolars seemed a good deal shorter than its fellows. There was no pain and tests for vitality were positive. As the phlebitis cleared up there was no dental treatment. Later a recurrence of the trouble led to further skiagrams and another premolar tooth had become shortened. Both these teeth were extracted and apical cultures gave a growth of a hæmolytic streptococcus. Further absorption of ' live ' teeth took place and the end-result was the extraction of all his remaining teeth, and subsequent vaccination with the hæmolytic streptococcus. Not until this was done did the phlebitis finally disappear.

The case suggests that the infecting streptococcus, which must have gained entrance *via* the grazed heel, had been carried to the teeth, where it manifested itself by eroding the apices. As the primary focus had long since healed the dental focus became all-important.[1]

A blood-borne infection derived from the jaws sometimes complicates the simple hæmatoma of contusions. It has already been mentioned that **puerperal fever** may occasionally arise in this way.

Some degree of **secondary anæmia** may result from hæmolysis due to streptococcal jaw infection, but Witts states that he has not found focal infection to be a common cause.

Since the elimination and detoxication of undesirable substances in the blood is performed by the kidney and liver, it is likely that prolonged absorption of toxic products damages these organs at last. Graham [2] was able experimentally to produce fatty degeneration in the liver and kidneys of animals by the injection of a sterile filtrate from human parodontal pockets. Clinically, not only acute but subacute and **chronic nephritis** appear to depend sometimes on a dental cause.

A medical practitioner of 45, who had served in the East during the war and had had dysentery, suffered in 1923 from vomiting and diarrhœa and swelling of the feet. Upon examination he was found to have severe nephritis with much albumen in the urine and a blood urea of 249 milligrams (normal about 36). His hæmoglobin was 35 per cent. and his teeth were in a very bad state. One tooth was removed every week and a long-chain streptococcus was grown from the roots of the teeth and the colon. It was found that about 30 hours after the removal of each tooth blood appeared in the urine and the patient had renal colic, with the eventual passage of blood-clots. This sequence of events was repeated sufficiently often to make us certain that the removal of each tooth caused a hæmorrhage, and suggested that the oral sepsis might have accounted for the nephritis.[1]

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[1] A. Bulleid, *loc. cit.*

[2] J. W. Graham, *Proc. Roy. Soc. Med.*, 1937, xxx, 1165.

(H) THE RHEUMATIC DISEASES.

The etiology of the rheumatic diseases is still somewhat obscure, but there is strong evidence that foci of streptococcal infection are one of the important factors. In these diseases the removal of such foci has caused more dramatic successes and more disappointment than any other form of treatment.

Acute rheumatism (rheumatic fever), a febrile disease of childhood characterised by acute inflammatory changes in the joints, heart, and other tissues, is strongly associated with streptococcal infection, most commonly an acute tonsillitis due to a *hæmolytic streptococcus*. The teeth are not frequently incriminated, though cases following acute alveolar abscess are on record. The condition is a very important contra-indication for dental operations. Owing to the simple endocarditis which is usually present, teeth should not be extracted during the acute phase of the disease, on account of the danger of setting up bacterial endocarditis (*q.v.*) ; and special precautions should be taken before dental operations at any time afterwards.

Rheumatoid arthritis, a general disease commencing commonly between the ages of 20 and 40 years, often with an acute onset, and characterised by inflammation about many joints both large and small, by fever, wasting, sweating, and other disorders, is strongly suspected to be principally of infective origin. It is in cases of this type that the best results have been obtained from the removal of septic teeth. The joint changes are at first peri-articular with synovial proliferation and some involvement of the epiphyses, but later the cartilage is destroyed by invading tissue, and fibrous or bony ankylosis may follow. Besides infection, principally streptococcal, the other factors in causation are individual constitution, endocrine, psychological and digestive disturbances, and occupational hazards. According to

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[1] E. P. Poulton, *Guy's Hosp. Gaz.*, 1933, lxxxiii, 137.

Willcox dental disease is the commonest primary source of infection. In certain cases the infective factor is much more prominent than the others, and these are sometimes grouped as 'infective' or 'secondary atrophic' arthritis. The suggestion is that septic foci produce toxins or antigens which pass continually into the circulation and cause this characteristic reaction in susceptible persons; but some think that an intermittent bacteriæmia is responsible. It should be noted that only an occasional organism is found in the joints: there is no secondary focus in the ordinary sense.

Many striking recoveries have followed the removal of dental infective foci, with immediate or progressive improvement and final cure; but the proportion of cases in which this occurs is unfortunately not large. The prospects are best during the early stages of the disease: when the cartilage has become seriously damaged the chances of considerable improvement are poor. When infective foci are present in the jaws it must not always be assumed that they are the only or most important focus. If the extraction of teeth be necessary they should be removed in small numbers after preliminary scaling and gum treatment. The liberation of antigens into the circulation caused by the operation may produce an acute temporary exacerbation of the rheumatic symptoms and make the patient feel intensely miserable or even do permanent harm. If the reaction after taking out one tooth be very severe, desensitisation with a vaccine prepared from this tooth may be advisable before others are removed. Even when no teeth are present the possibility of a focus in the alveolar bone must be considered.

Osteo-arthritis rarely occurs before 40 years and has an insidious onset. Here the patient often appears robust and has few symptoms apart from pain in one or more joints. There is synovial fibrosis, 'lipping' of the marginal epiphyseal bone, and degeneration of the cartilage, the last often disappearing entirely from parts where pressure is great. Ankylosis does not occur. In this condition there is much less evidence of an infective factor than in rheumatoid arthritis, tissue-age and trauma playing a great part. Many authorities, however, consider that infection is an important factor at some stage. Advanced parodontal disease is common in these patients, perhaps owing to the age-distribution of the affliction; but the prospects of great improvement following the removal of teeth are not very good, if for no other reason because serious destructive changes in the joints have usually already occurred owing to the insidious onset of the disease. A severe reaction to the removal of teeth is uncommon.

The relation of **fibrositis** and **interstitial neuritis,** including 'muscular rheumatism,' **lumbago, sciatica,** 'neuritis' and similar conditions, to infective foci in the mouth is similar to that of rheumatoid arthritis, and the same precautions in dental treatment are necessary.

(I) THE CENTRAL NERVOUS SYSTEM.

The suggestion of Cotton that focal infection plays a great part in the genesis of mental diseases has not been generally accepted. In the association of oral sepsis with mental illness the former is often an indirect result of the latter; and where improvement follows dental treatment it may often be due to suggestion or to the periodical intermissions common in many psychoses. In the **infective exhaustive psychoses,** however, which may be due to streptococcal infection, there is sometimes a direct connection with dental foci, and in these cases the treatment of infection leads to improvement or cure. The symptoms vary from neurasthenia and confusion to delirium and dementia. Operative treatment must be carried out with great caution, since a severe reaction is to be anticipated,[1] and must be avoided if possible. In all mental cases dental operations should only be performed in co-operation with the physician and at a chosen time, depressive phases, for example, being unsuitable.

According to some authorities infection from about the teeth may produce an ascending **neuritis** of the sensory branches of the trigeminal nerve with neuralgic symptoms. Other forms of interstitial neuritis have already been mentioned; and neurological effects produced by reflex action will be described in the next chapter.

(J) THE EYE.[2]

The importance of infection about the teeth as a source of disease in the eye has been definitely established, though it must be admitted at once that considerable revision of opinion has taken place since the day, some twenty-five years ago, when this idea was first seriously put forward. On looking back it is obvious that at that time the idea was accepted with uncritical enthusiasm ; it is possible that the pendulum has now swung a little too far the other way, and that the present tendency may rather be to neglect the eradication of distant focal sepsis in dealing with a case of inflammation of the eye. The pathology of eye-lesions is in many cases still obscure, and it should only be assumed that a causal relationship exists between the dental disease and the eye-lesions when (*a*) dental sepsis is undoubtedly present, and (*b*) the eye condition clears up with the removal of the dental sepsis.

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[1] A. Bulleid, *Brit. Dent. Journ.*, 1930, November 15.

[2] This section has been kindly rewritten by F. W. Law.

In a fair proportion of cases inflammatory disease of the eye is favourably influenced by the eradication of such foci. In general it is the more vascular parts of the eye, such as the conjunctiva, iris, ciliary body, and choroid, which are affected, rather than the less vascular sclera, retina, and optic nerve ; and histological evidence would seem to show that the cause is more often an intoxication than a secondary bacterial localisation, though the demonstration of bacteria in ocular tissues is notoriously difficult.

In any case of **conjunctivitis, iritis, iridocyclitis, cyclitis,** or **choroiditis** of which the pathology is obscure, it would be a serious omission in treatment if any septic dental focus were not dealt with. In some cases the effect is obvious and even dramatic, while in many it must be admitted that the result falls short of expectations ; and it must always be remembered that it is to the dramatic cases that publicity is given, a false impression being thus created as to the proportion of cases which may justifiably be expected to benefit from such treatment.

Cases of retrobulbar neuritis are sometimes reported as having benefited from the eradication of a focus of dental sepsis. While such a result does sometimes ensue, a report of this kind must always be accepted with reserve, since these cases almost invariably do improve whether treated or not. In the light of recent research it would seem that many of them must be attributed to disseminated sclerosis—a disease of which the local manifestations are characterised by spontaneous remission.

Most ophthalmic surgeons are agreed that it is advisable to eradicate obvious dental sepsis before an intraocular operation such as removal of cataract, although the investigation is rarely carried so far as to include a radiographic examination, and consists as a rule merely of a search for septic pockets about the teeth and gums. Such pre-operative preparation as is necessary should be undertaken a sufficiently long period of time before the operation to allow the systemic effects of dental extraction to subside, otherwise the result may be worse than that of leaving the dental condition undisturbed. Should, however, intraocular inflammation follow an operation which has been performed without previous attention to the mouth, and dental sepsis be suspected as a cause, such attention must be given at once in an endeavour to save the eye. It is seldom that serious exacerbation of an already existing eye infection is caused by dental treatment, though often a temporary flare-up is caused : in such cases the result of treatment is the more likely to prove ultimately beneficial.

While dental sepsis is a proved cause of ocular inflammation, it is never possible to promise a cure as a result of dental treatment to a patient who may, perhaps, be unwilling to submit to the loss of some or all of his teeth in the problematical interest of his eye.

The position is the same as when dealing with cases of fibrositis or sciatica or any other disease which experience has proved to be due in some cases to dental sepsis. Each case must be judged on its merits, and the clinician must preserve a balanced outlook. The soundest attitude consists in taking all precautionary pre-operative measures in order to avoid at least one proved possible cause of infection, and in cases of established disease it is necessary to remember the ever-present possibility of dental sepsis as a cause and to proceed accordingly.

(K) THE SKIN.[1]

The relation between dental infection and certain skin diseases has been accepted by dermatologists. The mechanism may be discussed under the following headings :—

1. By a nerve reflex from the teeth producing a reaction in the corresponding area of the skin (discussed on p. 817), giving rise to localised rosacea, and causing exacerbation of acne.

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[1] This section has been kindly rewritten by L. Forman.

2. Direct infection of the lips and adjoining skin by bacteria carried from the teeth by heavily infected saliva.

3. Absorption of organisms or their toxins from infected teeth into the blood stream may produce widespread inflammatory reactions of various types in the skin. These changes may be chronic or recurrent, depending on the response of the body and skin to such blood-borne infection, and on variations in activity of the focus of infection in the teeth. Further, the absorption of such bacterial toxins may modify the normal response of the skin to both external bacterial skin infection and to irritants of a chemical nature, *i.e.* may unfavourably influence the immuno-allergic reaction of the skin. This mechanism is operative in chronic staphylococcal infections and in some chronic eczemas of the hands and forearms in patients exposed to chemicals or soap solutions, etc.

4. The swallowing of infected saliva or actual pus may produce gastritis or at least interfere with the normal digestive processes, and modify the bacterial flora of the intestine. Absorption of toxic substances from the gut may embarrass the liver and adversely affect its normal detoxicating functions. Under such conditions, protein molecules capable of producing sensitisation of the skin may be absorbed into the blood stream.

Some of the dermatological conditions which may be caused or maintained by dental sepsis may be mentioned shortly :—

Cheilitis, an inflammation of the red of the lips, is often produced by infected teeth and gums. In the chronic forms there will be scaling and cracking, with a varying degree of swelling of the lips.

Intertriginous dermatitis at the angles of the lips, a persistent condition, is not relieved while there is dental sepsis, particularly of the open type. Similarly, patches of **chronic impetigo** on the chin or lips, with much oozing and crusting, are prolonged by constant contamination with infected saliva.

In various staphylococcal skin diseases dental sepsis is recognised as a causal factor. They include superficial **staphylococcal folliculitis,** particularly on the beard region ; **sycosis,** a very chronic confluent staphylococcal folliculitis of the beard region ; and **boils.** Here the resistance to staphylococci is lowered, and infected saliva continually reinoculates the skin with this organism. There have been many cases of chronic boils remaining resistant to treatment until dental sepsis has been treated; for example, in the case of a doctor who had been a martyr to boils for years, relief was only obtained after removal of infected teeth and alveolar bone.

Purpura, usually affecting the lower legs, may be caused by circulating toxins, derived from infected teeth, damaging the endothelium of the capillaries.

Recurrent erythemas, including **Erythema multiforme,** may have a similar etiology.

Lupus erythematosus, particularly of the localised type, has been improved or cured by the removal of dental sepsis.

Although psoriasis is a disease of unknown origin, the treatment of dental sepsis in some extensive cases has been accompanied by definite improvement.

There is a more direct association between dental sepsis and a pustular condition of the hands and feet, known as '**pustular bacterid.**' Eradication of sepsis in the teeth and tonsils leads to definite amelioration of the eruption.

Pruritus, prurigo, and **eczema** have been similarly relieved when dental sepsis has been treated. The influence of dental sepsis explains the rebellious character of some of these cases. Thus Darier notes that he has seen many eczematous patients cured of their incessant relapses by dental treatment.

Chronic urticaria and **angioneurotic œdema,** particularly in middle age, demand careful investigation and treatment of the teeth.

Rosacea is often accompanied by gastritis or dyspepsia, which may be secondary to dental sepsis.

(L) PRINCIPLES OF DENTAL TREATMENT.

With regard to the prevention of the remote effects which have been mentioned, the counsel of perfection is that teeth must not be allowed to decay or parodontal disease to develop. When these things have occurred, however, radical local treatment carried out with care will prevent damage to other parts of the body. The difficulty is that there are often objections to radical treatment on æsthetic or functional grounds. The extent to which conservative treatment is justified must depend on the nature and extent of the oral lesions and on the age and constitution of the patient. Risks may be taken with the young and vigorous which are quite unjustified in the case of the middle-aged or weakly.

When symptoms or diseases which may be due in part or entirely to infection about the teeth are already present, or where serious operations are contemplated, a conservative policy should only be followed if there is good reason to suppose that it will be efficient, as in the case of early parodontal disease or dental caries which has not affected the pulp. Otherwise, removal of the infected teeth or surgical drainage of the infected area is indicated, but such treatment should always be carried out in co-operation with the patient's physician at the most appropriate time. It has already been said that it is usually quite impossible without experiment to be certain whether remote symptoms are due to a dental focus or not, even when no other cause can be found. But as there is evidence that the course of nearly all diseases is adversely affected by concomitant oral infection, whether they be caused by it or not, the removal of a source of infection is always desirable.

The greatest difficulty arises in cases where remote symptoms are present, and of a kind which may be due to infective foci in the jaws, and where infection about the apices of certain teeth possibly, but not certainly, exists. In such a case it is reasonable to weigh the importance of the remote symptoms, including their possible extension, against the disability or disfigurement which may be caused by the loss of the teeth in question. Sometimes the removal and bacteriological examination of one tooth will provide an indication of the state of the others, and the reaction to this small operation may also give information. Where a minor degree of parodontal disease is present, modified conservative treatment by gingivectomy may be employed. According to Fish,[1] even in more advanced cases where the teeth affected by the disease are still of functional value, parodontal infection can be eradicated by gingivectomy, provided that the pockets are not too deep and that the patient is prepared to co-operate in keeping the gums healthy afterwards ; and the teeth may then be retained without danger.

Teeth should not be removed unless the dental surgeon is satisfied that their removal is necessary for the suppression of foci of infection in their neighbourhood. Any benefit which results from the removal of sound teeth is due either to suggestion or non-specific shock, and both forms of therapy can be applied better in other ways.

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[1] E. W. Fish, *loc. cit.*

On the other hand, it goes without saying that the dental surgeon should never declare the mouth healthy until he has examined good radiographs which include the edentulous areas.

When the extraction of teeth is necessary the teeth should first be scaled and superficial tissues should be made as clean and healthy as possible. The use of the cautery has already been mentioned. The extraction of one tooth only on a subsequent occasion is a wise proceeding in all serious cases, and the reaction provoked should be noted. It is always prudent for the dental surgeon to inquire from the physician before the first tooth is extracted whether it is desired that a bacterial culture shall be made or not. The physician may be disgusted to find that a tooth from which he planned to prepare a vaccine has been cast in the dustbin, even though he may have made no mention of his intentions beforehand. An interval of five or six days should pass between the removal of successive batches, and the number taken at one time may be increased according to the strength of the patient. In the case of very feeble patients the number should not exceed four or five, as the consequences may sometimes be disastrous, but robust patients showing little reaction can be treated with less caution.

The very rapid progress of chemotherapy in recent years justifies the hope that it will be possible to counteract the dangers of bacteriæmia following dental operations by the use of sulphonamide and allied compounds. While undoubtedly successful in the case of most hæmolytic streptococci they are at present much less effective where *Streptococcus viridans* is in question. Their use is not free from danger and patients to whom they are administered should be under medical supervision.

CHAPTER XXVI

Diseases arising from Reflex Irritation from the Teeth.

Affections of the Nervous System—Affections of the Ear—Affections of the Eye—Affections of the Skin.

In past years various lesions of the eye, ear and other organs were attributed to reflex irritation from the teeth. In reading the accounts of many of these cases, one is driven to the conclusion that the diagnosis was often incorrect, and that diseases which were merely coincident were frequently wrongly associated in a causal relationship. The histories of the cases, when read by the light of modern pathology, would suggest that, in the majority of the cases, the lesions attributed to the teeth were the result of septic absorption rather than reflex irritation.

(A) AFFECTIONS OF THE NERVOUS SYSTEM.

Epileptic and Convulsive Seizures.[1]—It is possible that epilepsy may be induced by dental irritation. Brown Sequard found that, after section of one of the lateral columns of the cord anywhere between the medulla and the tenth dorsal vertebra, epilepsy could be induced by very slight irritation of the fifth nerve. The following case is recorded by Bakewski [2] :—

A young female had suffered from epileptic seizures for nine months, the fits having increased in frequency, there being several every day. The usual drugs were tried without effect. Finally, the teeth were examined and the maxillary right first molar and the mandibular left first molar were found to be carious. These were removed and the fits ceased entirely and did not return, the patient being kept under observation for six months subsequently. Upon being closely questioned, the girl remembered that before the fits commenced she had had some unpleasant sensations in the affected teeth, but nothing that could be described as pains.

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[1] The cases recorded under this and subsequent sub-headings have been introduced in order that the student may gain some idea of the evidence on which the relationship between reflex dental irritation and other diseases is based.

[2] *Brit. Dent. Journ.*, xii, 280.

Roland Still[1] records the case of a boy, aged 10, in whom epileptiform fits occurred, commencing about the age of 7 and gradually becoming more frequent and severe; there was no hereditary tendency to fits. There was extensive caries, oral sepsis, delayed and incomplete eruption of some teeth, and crowding and impaction in the mandibular incisor region, so that ten teeth were extracted on account of caries and two for impaction. No further fits occurred so long as the boy remained under observation.

A case of some interest came under the observation of Morton Smale :—

> A patient, aged 22, a male, always showed distinct signs of *petit mal* during the operation of filling, the symptoms being always more marked if an exposed nerve were touched. This patient, to the best of his recollection, had never suffered at any time with epilepsy, nor had he had fits of any other character.

E. C. Wilkerson[2] records the case of a lady, aged about 21, with pain in $|\overline{8}$ region and a history of *petit mal* over a period of two years; the gum over this tooth was incised and the frequency of the attacks lessened till a year later, when pain occurred in $_{\overline{8}}|^{\underline{8}}$ regions; incision of their overlying gum then gave entire freedom from attacks for ten months, when they again became frequent, this time synchronising with pain in $\underline{8}|$ region. Ultimately on removal of these teeth there was complete freedom from further attacks.

With persons subject to epilepsy it is not uncommon for a fit to be induced by the irritation attending dental operations.

Habit Spasm.—Some medical writers are of opinion that habit spasm may occasionally be connected with dental irritation. This is a functional condition, and it is possible that a dental lesion may induce it, either directly by reflex irritation or indirectly by lowering the vitality. The following case, quoted in the 'American System of Dentistry,' would seem to indicate that the dental trouble was intimately connected with the spasmodic condition, but it is unlikely that true chorea is ever the result of dental disease. The case is related by C. N. Pierce[3] :—

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[1] *Brit. Dent. Journ.*, lxiii, 468.

[2] *Ibid.*, 430.

[3] *Ibid.*, iii, 598.

A boy, aged 9, had always been in good health until two years ago, when he was attacked with choreic movements chiefly in the muscles of the face, though present also in the muscles of the neck and shoulders. Owing to their local character, and the absence of the causes usually assigned for the appearance of chorea, it was thought possible that the condition of the teeth might offer some explanation of the trouble. Careful examination of the mouth revealed, in addition to considerable overcrowding of the teeth, persistence of the deciduous incisors. Upon their removal the choreic movements at once subsided. After an interval of a year there was a return of the symptoms. Examination of the teeth again showed a persistence of the deciduous molars delaying the eruption of the premolars. Removal of the offending teeth was followed by complete recovery.

Spasmodic Closure of the Jaws.—The following case was reported by Ewart[1] :—

The patient, a man, aged 47, moderately addicted to alcohol, was suffering from an extensive chronic ulcer of the right leg. His illness began with a 'severe cold' three weeks before his admission into St. George's Hospital on September 20, 1899. The trismus set in quite suddenly during the night a week later, when he awoke in a fit of suffocation due to the closure of his lips unsupported by teeth. The same nocturnal attacks continued to occur in the hospital for five weeks. The rigidity of the jaws, of the floor of the mouth, of the platysma of the neck, and of the abdominal muscles was intensified by excitement, but there was neither opisthotonos nor any spasm of the limbs. Speech, respiration, and alimentation were much impeded, leading to loss of flesh and weakness. There was a tender and slightly swollen spot on the gum, and the case was diagnosed from the first as one of reflex spasm due to periosteal irritation and tenderness at the left posterior extremity of the upper jaw, and local treatment was recommended. This was finally resorted to, after various remedies had proved ineffectual, at the end of October, and the symptoms rapidly disappeared after the tender gum had been freely incised.

This case was probably purely reflex, the edentulous and healthy condition of the gums excluding the theory that septic infection from the mouth was the cause of the spasmodic closure of the jaws.

Many cases of inability to open the jaws arising from the teeth are not due to spasm of the masseter muscles (trismus) as is often supposed. The inability to open the jaw in most of the cases arises from infiltration of the muscles and adjacent tissues, the interference with the movement of the jaw being due partly to mechanical impediment from the inflammatory products and partly to involuntary closing of the jaw.

Paralysis of the Arm.—In the chapter on odontalgia reference was made to the occurrence of pain in the shoulder traceable to the teeth. It is interesting to note that cases of supposed paralysis of the arm have been recorded as due to dental lesions. Salter [1] records the following :—

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[1] *Brit. Journ. Dent. Sci.*, 1900, January 11.

> Miss B., aged 24, consulted me on October 15, 1864, respecting the mandibular left third molar, and the symptoms to which it had apparently given rise. The tooth had pierced the gum, but it was low down and placed horizontally, the crown pressing forwards against the second molar. The tooth was carious. From the first attempt at the eruption of this tooth there had been much pain of the ordinary kind about the angle of the jaw ; latterly it had been intense, and for a fortnight there had been paralysis of the left arm ; the patient complained of total inability to use the arm, to raise it or grasp with the hand ; she could not employ the limb in dressing herself, and could not hold her fork at dinner. There was also a continuous pain of the whole arm resembling rheumatism. The tooth was extracted, but with extreme difficulty. As soon as the patient recovered from the pain of the operation she declared that the arm symptoms had vanished completely.

J. H. Mummery records [2] that whenever he suffered from much pain in a mandibular left molar he could raise his arm only with great difficulty and experienced a sense of weight and fatigue in the arm almost amounting to pain.

In Salter's case, as in many similar records, no electrical examination of the muscles could be recorded. Before arriving at a diagnosis of paralysis it is always advisable to exclude hysteria, and to satisfy oneself that the case is not one in which the part is involuntarily kept inactive in order to avoid pain.

Spasm of the Sternomastoid (Wry-neck).—A case of spasm of the sternomastoid has been recorded by Hancock.[3] The patient, a woman, had suffered considerable pain in the left shoulder, apparently owing to the presence of a carious tooth on the left side of the mandible. The head was drawn down almost to the left shoulder. On removal of the tooth the condition disappeared. In this case and in other similar cases recorded it is probable that the head was involuntarily bent down as a result of the pain and that there was no true spasm of the sternomastoid.

Tonic Spasm of the Upper Extremities.—In the following case the spasm of the muscles would seem to have been related to pulp irritation :—

> A robust, strong man of 29, who was not in the least nervous, suffered from toothache, caused by an exposed pulp in the maxillary left third molar. During repeated attempts at luxation of the tooth, which was fixed very firmly,

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[1] 'Dental Pathology and Surgery,' 1874, 264.

[2] *Brit. Med. Journ.*, September 9, 1905.

[3] *Lancet*, 1859, 80.

the forceps slid off and struck directly into the pulp. There was at once a tonic cramp of the flexor muscles of all fingers on both hands. Especially the left hand was closed so tightly that the patient could not open it, whilst he had some difficulty in opening the right. The flexibility of the arm was also affected. The patient, describing his condition, said he felt as though a very strong electric current passed through his head and arms, or as if the latter had pins and needles in them; a painful condition which caused the patient, usually showing great powers of endurance, to groan pitifully. Concentrated carbolic acid was applied to the pulp, whereupon the cramp gradually ceased. On the tooth being extracted the patient became normal, and could follow his business undisturbed all day long.[1]

Paroxysmal Dyspnœa.—W. L. Balendra [2] records the case of a Singhalese woman aged 32. At the age of 22 she had suffered from asthma for eight months; it subsided with medical treatment. Ten years later the 'asthma' recurred, the spasms lasting about three minutes and being more frequent at night. No pain was referred to teeth and the case did not respond to medical treatment; the patient lost a stone in weight in six months. Radiologically $|\overline{8}$ was found to be horizontally impacted. $|\overline{78}$ were removed under general anæsthesia, and the asthmatic attacks ceased almost at once and remained absent during the ensuing two years that the patient remained under observation. She regained her normal weight in two months.

(B) AFFECTIONS OF THE EAR.

Otalgia.—Earache, due to reflex irritation from the teeth, is not uncommon.

Parodontal disease or a carious tooth will frequently infect the mucosa of the naso-pharynx and cause symptoms in the middle ear, of which the most common is earache. Politzer states [3] that carious teeth are the most frequent cause of earache in children. In the light of modern experience, this cannot now be accepted. Much the most common cause is the presence of acutely infected tonsils, the presence of adenoids, or an acute naso-pharyngeal infection, either catarrhal or pyogenic.

Much the commonest cause of earache, of dental origin, is the presence of an unerupted or erupting mandibular third molar, particularly when they are misplaced, and in all cases of earache, where there are no definite signs in the drum or the middle ear to account for the pain, a careful examination of the teeth should be made.

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[1] This case was recorded by G. Randorf in *Items of Interest.*

[2] *Brit. Dent. Journ.*, lx, 339.

[3] Edition 1909, 686. Translated by Messrs. Baillie and Heller.

Even when the ear symptoms appear to be sufficient to account for the otalgia, the possibility that the condition of the teeth may aggravate the pain should not be overlooked. Similarly, pain may be referred from the ear to teeth.

Deafness without definite lesions in the ear is at times apparently traceable to reflex trouble from an unerupted mandibular third molar. In one case a patient developed what she termed a ‘ worry ’ in the ear, which interfered with hearing. The ear was free from organic disease, and in the mouth the only possible cause of the trouble was an unerupted misplaced third molar. With the eruption and removal of this tooth the symptoms passed away. J. Howard Mummery [1] records a case of considerable deafness on the left side, which had existed for months in association with the delayed eruption of the maxillary third molar. The hearing was much relieved immediately after the extraction of the tooth and was fully restored the same day.

(C) AFFECTIONS OF THE EYE.

According to S. E. Whitnall,[2] reflex neurosis, from disturbance of the integrating mechanism of the fifth nerve, may lead to muscular disorders, twitching or pain and discomfort around the eyes, and sensory or vasomotor disturbances. He states that Black [3] considers these to arise nearly always from pulp diseases, but occasionally from impactions.

Lachrymation.—Apart from an emotional flow of tears from the distressing pain of odontalgia, the eyes will occasionally become suffused with tears through sudden pain caused by biting on a sensitive tooth. This is similar to the reflex overflow of tears produced by pungent vapours, *e.g.* strong ammonia.

Spasm of the Orbicularis palpebrarum (Blepharospasm).—Excessive blinking is a mild variety of chronic spasms of the orbicular muscle of the eyelids. In a case of this kind seen by Knies, the spasms subsided immediately after the removal of a painful tooth.

Blepharospasm is occasionally traceable to reflex dental irritation, and in such instances the trouble can be cured by attention to the teeth. Blepharospasm may, however, occur in elderly, edentulous people, and a cure is then difficult, if not impossible. In these cases it is supposed that the osseous sclerosis present causes the spasms reflexly by the imprisonment of filaments of the dental nerve. The spasm involves, in addition to the orbicularis palpebrarum, many of the other muscles supplied by the facial nerve, and varies from a slight twitching of the orbicular muscle to a violent spasmodic contortion of the whole side of the face. Blepharospasm is usually asymmetrical, and is mostly seen in elderly females. It has no association with *tic convulsif*.

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[1] *Brit. Med. Journ.*, 1905, September 9, 553.

[2] *Brit. Dent. Journ.*, lxiii, 412.

[3] *Ophth. Record.*, 1915, 24, 610.

Strabismus.—A squint not infrequently develops during the period of the eruption of the deciduous molars, and many mothers are convinced that the dental irritation causes the squint. In these cases the disturbance created by the eruption of the teeth may act as the determining cause of the strabismus, the fundamental cause being weakness of binocular vision and disordered muscle balance co-ordination.

Spasm of Accommodation.—Spasmodic contraction of the ciliary muscle, similar to spasm of the internal rectus, seems occasionally to have a reflex dental origin. Attention to the teeth will sometimes cause a chronic ciliary spasm to relax, and thus cure a troublesome case of apparent myopia.

Paresis of the Levator Palpebræ Superioris.—Hancock[1] and Nicol[2] have each recorded a case of ptosis which disappeared after the treatment of carious teeth. In all probability the ptosis in these cases was purely functional.

Muscular insufficiency with diplopia has been attributed to painful carious teeth.

Paresis of Accommodation.—Accommodation failure, attended by asthenopia, is recognised by many surgeons to be due to diseases of the teeth. Schmidt[3] found it present in seventy-two out of ninety-two cases of dental caries. It probably results, as Knies[4] has suggested, from the lack of vigorous innervation brought about by the distressing pain. On the other hand, Priestley Smith found unimpaired accommodation in fifteen out of sixteen cases of odontalgia.

Amaurosis and Amblyopia.—Amaurosis, or complete functional loss of sight, and amblyopia or impaired visual acuity, are two functional conditions which have repeatedly been attributed to reflex dental irritation. There are many such cases on record. In some of them there was probably an acute retrobulbar optic neuritis, the result of inflammation by continuity of tissue, while in others the elements of hysteria probably predominated. Galezowski records a case in which a lady suffered from impaired vision after a tooth had been filled; her vision improved on the removal of the filling and relapsed again when the tooth was refilled. The removal of the tooth wrought a permanent cure.

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[1] *Lancet*, 1859, 80.

[2] *Trans. Odonto. Soc.*, November 1895.

[3] *Arch. f. Ophthal.*, xiv, 107.

[4] 'Relation of Diseases of the Eye to General Diseases,' 267.

The following case of amaurosis consecutive to the extraction of a tooth is recorded by Santamaria (*La Stomatologia*, Milan, July 1906).

> The patient was a soldier who, prior to enlisting, had followed the occupation of portrait painter. Six months after his arrival at the Florence barracks he suffered from a violent attack of inflammation of the pulp in the maxillary right permanent first molar, which tooth, being decayed, was extracted at once. A few hours after the extraction the patient again suffered from a neuralgic attack, the pain radiating from the right cheek to the eye of the same side, and from a rapid and progressive diminution in the visual power of the right eye, which very soon became amaurotic. This condition lasted five days, and, ceasing abruptly, was followed by intense amblyopia. An examination showed that the visual field had decreased both on the right and left sides, but to a greater extent on the former than on the latter, and that there was, in addition, an intense dichromatism for the blue and red primary colours. An external examination revealed nothing worthy of note; in the mouth no pathologic lesion could be seen.

The conclusions regarding the peculiarities of this case are that the amblyopia was not of reflex nature; that the dichromatism was an interesting feature of the case by virtue of the previous occupation of the patient; that the amaurosis and the dichromatism confirm the fact observed by Trombetta, viz. that the symptoms exhibited by those affected with traumatic neurosis become in preference localised in those organs which for each patient are of pre-eminent importance in his daily occupation; that the nature of the traumatism was entirely out of proportion to the severity of the consecutive neurotic manifestations, and that even the most insignificant stimuli may be followed by serious and unexpected consequences in the case of neuropathic patients.

Glaucoma.—In referring to strabismus it was pointed out that the irritation from eruption might in some cases be the determining cause; in like manner it is possible that odontalgia may be the determining cause of a glaucoma. If an eye is anatomically in a condition favourable for the onset of an attack of glaucoma, severe pain arising from a carious tooth may so lower the vitality of the patient that the disease may be started. Von Hippel and Grünhagen [1] consider that irritation of the fifth nerve raises the intraocular pressure. Priestley Smith's [2] experiments, however, seem to show that the tension is not increased in odontalgia.

Defects associated with Unerupted Third Molars.—Bowdler Henry [3] states that healthy but unerupted third molars may produce serious ocular disturbance either by direct pressure on nerves or by reflex irritation. He quotes cases of blepharospasm, muscle paresis, ptosis, proptosis and monocular mydriasis cured by removal of misplaced third molars; and cases of refractory uveitis and ocular pain cured by X-rays and removal of buried third molars.

(D) AFFECTIONS OF THE SKIN.[4]

Clinical evidence suggests that certain skin eruptions may be provoked by reflex irritation from the teeth as a result of caries, impaction, or metallic fillings. Attention has been drawn to the latter recently since it has been shown that electrolytic phenomena can be demonstrated when amalgam fillings of different types are present, and a number of cases of chronic or recurrent ulcers of the tongue or cheeks of obscure etiology have cleared up after removal of the metal. Probably some of the recurrent ulcers were herpetic.

Herpes simplex of the recurrent type is probably the most common eruption due to reflex dental irritation, and recently Silcock described a case of it affecting the cheek and recurring in spite of treatment until metallic fillings were removed with immediate effect. Although herpes is due to a virus the eruption is often determined by some stimulus to the nerve or to the skin or mucous membrane it supplies, corresponding to a ganglion in which the virus presumably resides.

Herpes zoster less commonly affects the face and usually requires a more drastic stimulus to provoke it, such as dental extraction with unusual trauma. It often occurs spontaneously and rarely more than once, so that it would be impossible to estimate the causative effect of coincident dental irritation.

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[1] *Arch. f. Ophthal.*, xiv, 1, 107.

[2] Priestley Smith, 'Glaucoma,' London, 1879.

[3] *Brit. Journ. Ophthalmol.*, 1935, 378.

[4] This section has been kindly rewritten by Reginald T. Brain.

Alopecia areata frequently appears to follow shock and anxiety states, but only rarely are local foci of irritation discovered in areas supplied by the cranial nerves. A thesis was once put forward that areas of baldness in alopecia could be correlated with segments of the scalp and caries in certain teeth, but observations by Sequeira on a further group of patients gave entirely negative results, and a dental factor is not recognised as important in alopecia, with the exception that caries or pyorrhœa may account for a toxæmia affecting hair growth.

Acne rosacea has a reflex factor more often in the nose than in teeth ; in fact, the latter must be very rare or overlooked. More commonly neglected caries results in chronic gastritis, and this is much more important in rosacea.

Acne is unlikely to be dependent upon reflex irritation, for it has endocrine and constitutional factors, and the disturbance of sebaceous gland function is widely spread.

Angioneurotic œdema and similar erythematous or œdematous lesions affecting the face, buccal or pharyngeal mucosa may be due to reflex irritation of dental origin, but so often they are not that only a dramatic response to some dental operation would suggest the relationship.

CHAPTER XXVII

The Operation of Extraction and the Surgical Removal of Teeth.

The General Principles of Extraction—Extraction of Individual Teeth and Roots—Extraction of Misplaced Teeth—Extraction of Teeth under General Anæsthetics—Difficulties, Complications, and Sequelæ of Extraction of Teeth.

Extraction is an operation which requires skill, judgment, experience, and an accurate anatomical knowledge of the parts involved. As with all other manipulative proceedings, success can only be attained by actual practice.

(A) GENERAL PRINCIPLES.

The tooth to be removed should be carefully examined in order that the operator may gain some idea of the amount of sound tissue present and the degree of force which will be necessary to dislodge the tooth. In the case of roots the edges must be defined, and for this purpose a blunt probe will be found useful.

(1) Instruments.

The instruments in general use for the removal of teeth are forceps and elevators.

The forceps is made up of three parts, namely, the blades, or portions beyond the joint, which are applied to the tooth, the joint itself, and the handles. Forceps should be made of fine steel, light, and yet strong enough to withstand without bending any strain that it may be necessary to put upon them. Blades shaped to fit the tooth they are intended to remove should be used, and when applied they should be clear of the crown. On longitudinal section a blade should present a thin wedge-shaped appearance.

The handles should be of a size and shape to lie comfortably in the palm of the hand, and their relation to the blades should be such as to clear the lips when the blades are applied in the direction of the long axis of the tooth. For the anterior maxillary teeth the blades and handles should be in the same line (fig. 832); for the posterior maxillary teeth the blades should form a curve with the handles (figs. 833 and 834); for the mandibular teeth the blades should be bent down from the handles at an angle of ninety degrees (figs. 835 and 837).

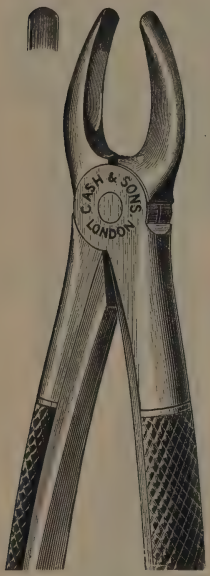

Fig. 832.

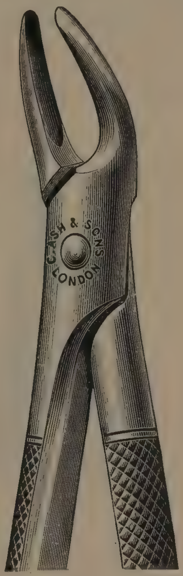

Fig. 833.

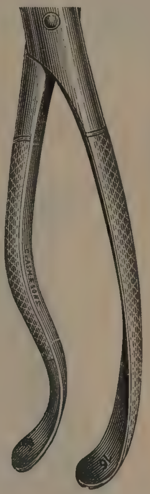

Fig. 834.—Suitable handles for all forceps for extracting maxillary teeth.

(For the use of the blocks illustrating the various instruments we are indebted to The Amalgamated Dental Company Ltd.)

For mandibular teeth and stumps many operators prefer to have a thumb rest as in fig. 836; it gives additional purchase and ease in exerting downward pressure in the application of the beaks.

The elevator consists of two parts—the handle and the blade. The handle is about four inches in length and of a shape suitable to allow a firm grip to be obtained. The blade is of fine steel and about two inches long. Elevators are either straight or curved. In the straight variety the blade is thin, about one-fifth of an inch in breadth, one surface being made convex and the other flat; the point of the blade should be rounded as shown in fig. 841. In the curved variety, the terminal half-inch of the steel part of the instrument is bent at an angle with the shaft of the instrument (fig. 858). The edge of the blade of an elevator should always be kept sharp.

The screw (fig. 842) is an instrument which on rare occasions is useful for the removal of deep-seated roots.

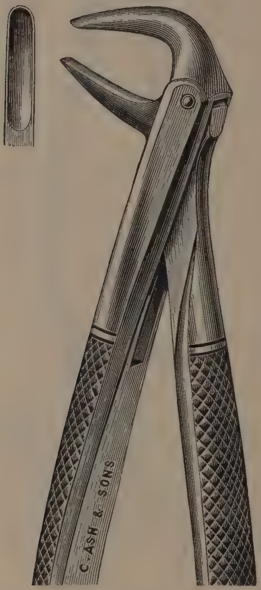

FIG. 835.

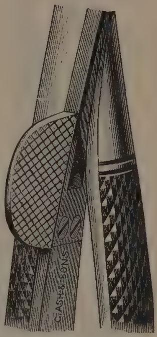

FIG. 836.—Thumb rest on handle often used with fig. 835.

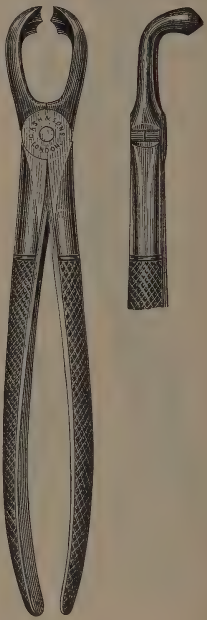

FIG. 837.

(2) Method of Holding Instruments.

Forceps should be held as shown in figs. 838 to 840. The handles should rest comfortably on the palmar surface of the hand, and in such a manner that the end of one handle rests between the thenar and hypothenar eminences—the part of the hand where force can be applied with the greatest advantage.

The thumb placed between the handles acts as a regulator to control the pressure of the blades upon the tooth. As a precaution it is well to have the ball of the thumb well between the handles,

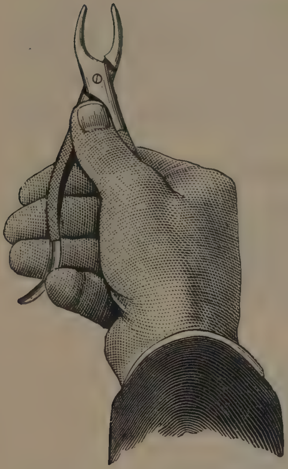

FIG. 838.—Mode of holding forceps for the removal of maxillary teeth.

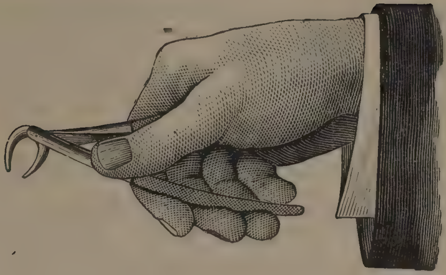

FIG. 839.—Mode of holding lower 'hawk's bill' pattern forceps.

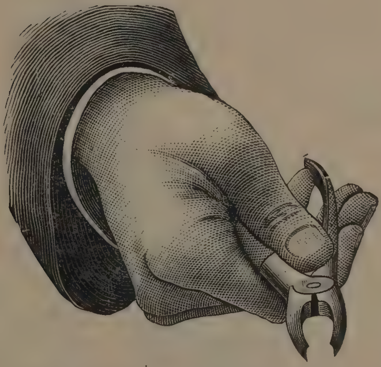

FIG. 840.—Mode of holding forceps of pattern shown in fig. 837.

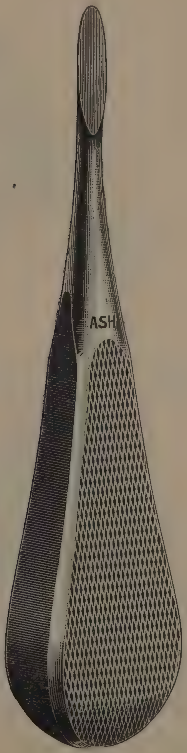

FIG. 841.

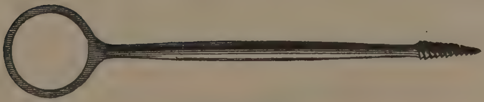

FIG. 842.

so that the pressure is counteracted by the terminal bony phalanx of the thumb as well as by the soft tissues. If this precaution be not observed, any sudden crushing of the tooth may be accompanied by a severe and very painful contusion of the operator's thumb.

The method of holding an *elevator* is shown in fig. 843. The handle should rest comfortably in the hand, the first finger lying along the blade and being brought near the point so as to prevent the instrument from slipping. The first finger should lie along the curved side of the blade for extractions on the right side of the mandible, and along the flat side of the blade for extractions on the left side of the mandible.

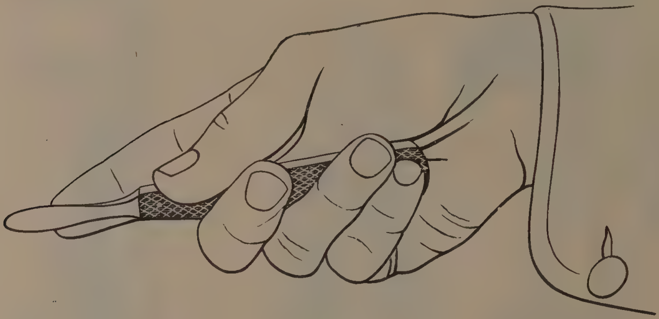

FIG. 843.

(3) Position of the Operator and the Patient.

There should be a good light in front of the operating chair, and, if a proper dental chair is not available, an ordinary armchair may be utilised. The patient should be placed in an unconstrained position, but in such a position as will allow the operator all the freedom of movement which he may require.

The position of the operator should be such as to enable him to use force to the greatest advantage. His left arm may be utilised, if necessary, for steadying the movements of the patient's head, while the fingers of the left hand can be employed :—

(*a*) To control the cheek and other soft parts so as to obtain a clear view of the tooth to be extracted and its immediate neighbours.

(*b*) To support the mandible.

(*c*) To grasp the alveolar process in order to gain some idea of the effect of the force employed.

The special positions for the removal of different teeth are described on p. 829.

(4) Anatomy of the Teeth and Jaws.

If the teeth be examined it will be noticed that as regards the roots they fall into three divisions :—

(*a*) Teeth with single, round, tapering roots.

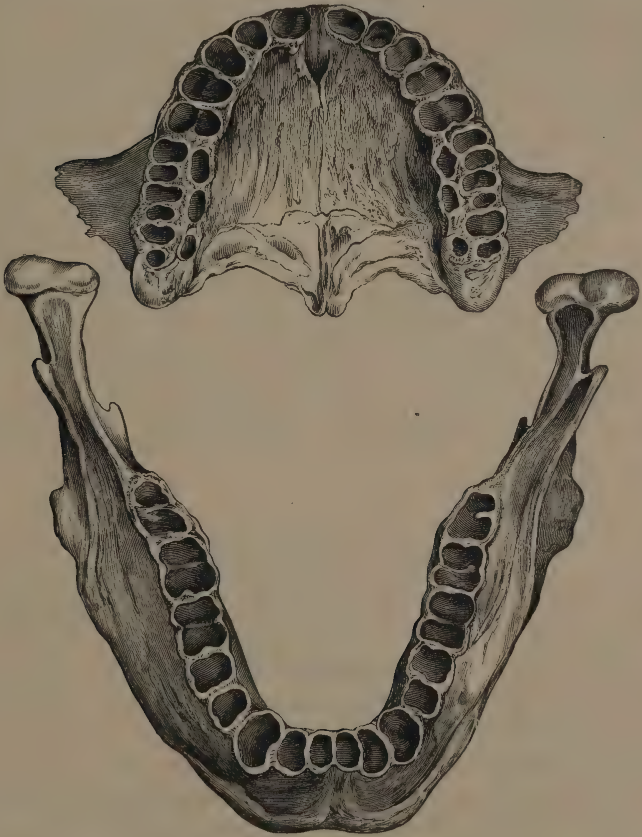

Fig. 844. (From 'The American System of Dentistry.')

(*b*) Teeth with single roots which are more or less irregularly flattened or curved.

(*c*) Teeth with multiple roots.

Division (*a*) comprises the maxillary first incisors (deciduous and permanent), the mandibular premolars.

,, (*b*) The mandibular incisors and canines (deciduous and permanent), the maxillary canines and second incisors (deciduous and permanent), the maxillary premolars.

,, (*c*) The maxillary and mandibular molars (deciduous and permanent).

The shape of the roots, as we shall subsequently find, has an important bearing upon the manner in which force is to be applied when severing the roots from their attachments. To ensure skilful and successful operations a thorough knowledge of the alveoli of the teeth is necessary, and it is needless to say that this knowledge can only be gained by careful study of the bones (fig. 844). Some idea of the strength of different portions of the alveolar border will thus be obtained, a matter of some moment when force is being applied in the process of removing a tooth from its socket. In the maxilla the points to be specially noted are the thinness of the outer alveolar wall as compared with the inner, and the prominence of the bone in the region of the third molar. In the mandible, the outer alveolar border is, it will be seen, thinner than the inner, with the exception of that portion in the region of the third, and often of the second, molar ; at the posterior portion of the socket of the third molar the bone is moderately dense.

(5) Method of Using Instruments.

The operation of tooth extraction when performed with forceps may be divided into three stages [1] :—

(*a*) Adjustment of the forceps to the tooth.

(*b*) Rupture of the membranes connecting the tooth with the socket and dilatation of the socket.

(*c*) Removal of the tooth from the socket.

In the initial stage *the first step* is the application of the blades. Care must be taken that the points pass between the gum and the tooth, and also that the blades are applied parallel with the long axis of the root.

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[1] A description of the form of each tooth, with respect to its bearing upon the construction of forceps for its removal, will be found under the heading (B).

As a rule it is best to apply one blade first on the side of the tooth most obscured from view, and then lightly to close the other upon the opposite side. The blades should then be forcibly pressed upwards or downwards, as the case may be, in the direction of the apex of the root; a slight rotary or wriggling motion will often be found of assistance in the process. This 'pressing' movement should be continued until a firm hold of the root has been obtained—a matter of great importance, as the successful removal of the tooth depends in a large measure upon the grip obtained. The handles should next be closed so as to give the blades a firm grip, and the amount of pressure applied should be such that when movement has commenced the blades do not ride upon the surfaces of the root. The amount of pressure must of course depend upon the character of the tooth to be removed and the resistance offered by the alveolar process. The thumb placed between the handles of the forceps, as already explained, should counteract the pressure on the root and prevent crushing.

The second stage—the rupture of the membranous attachments and dilatation of the socket—is accomplished by employing force in either a rotary or a lateral direction. The movement to be employed depends upon the form of the root or roots to be removed and the resisting strength of the surrounding hard structures. Rotary movement is of course admissible only in the case of teeth possessing a single conical root.

The final stage is carried out by exerting extractive force in the direction of the long axis of the tooth, while at the same time following the line of least resistance; the latter is determined by a knowledge of the anatomy of the alveolar border and by the sensation conveyed to the hand through the forceps.

Elevators.—*The removal of a tooth with a straight elevator* is accomplished in the following manner:—

The blade, with the flattened surface towards the tooth to be removed, is inserted between the root and the alveolar process, this surface being kept as far as possible parallel with the anterior surface of the crown. The blade is then forced downwards so as to reach the root at a point as low as possible; the handle of the elevator is then rotated away from the direction in which the tooth is removed. This has the effect of raising the tooth in its socket and at the same time displacing it in the required direction. One such movement of the instrument rarely suffices for the removal of a tooth, and a second, and sometimes a third grip, each time nearer the apex of the root, has to be obtained.

The method of using a *curved elevator* will be described in dealing with the removal of the roots of mandibular teeth.

The Screw.—Before attempting to remove a deep-seated root with the screw, a good view of the root should be obtained by pressing the overlying tissues away. A clear view of the root canal having been gained, the screw should be carefully worked into the canal, and when sufficient hold has been obtained extractive force should be applied.

(6) The Preparation of the Mouth.

The tooth to be extracted should be freed from all foreign matter by the removal of any adherent salivary calculus and the thorough irrigation of the space around the teeth. For the purpose of irrigation peroxide of hydrogen is most useful, and the mouth should also be thoroughly rinsed with a suitable wash. The soft tissues around the tooth to be extracted should be swabbed with a 2 per cent. solution of iodine in alcohol. When many teeth have to be removed at one sitting every endeavour should be made to render the mouth as aseptic as possible before operating, especially in the case of patients suffering from parodontal disease. It is a good plan to prescribe a mouth-wash to be frequently used for at least one week before removing the teeth. The patient should also irrigate the individual sockets with a suitable antiseptic. By the removal of the sepsis in the mouth the resistance of the tissues is raised and they can react more readily to the injury caused by the removal of the teeth.

(7) The Treatment of the Wound.

The wound resulting from the removal of a tooth is a lacerated one. It is best treated by keeping the parts carefully cleansed from all foreign matter, and for this purpose an antiseptic mouthwash [1] should be used several times a day. On the extraction of

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[1] The following is a useful formula :—

℞	Liquoris potassæ	ʒvi.
	Acidi carbolici glacialis	ʒiv.
	Aquam ... ... ad	℥iv.

M. One teaspoonful to be used with half a tumblerful of warm water as a mouth-wash.

a maxillary tooth the discharge from the wound drains away naturally owing to the position of the orifice. The wound caused by the removal of a mandibular tooth cannot drain naturally and, should sepsis be present, the socket must be kept clean by gentle syringing with some antiseptic solution.

The repair of the wound caused by the extraction of a tooth has never been carefully studied, but there is no reason to suppose that the process differs from that seen in the repair of other bones. The changes in the bone as seen in dried specimens are as follows :—

(1) The margins of the sockets undergo slight absorption (fig. 845, *a*).

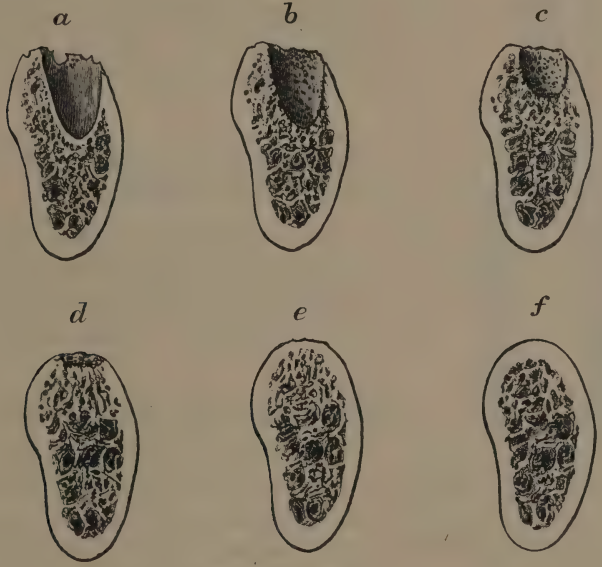

Fig. 845.

(2) The wall of the socket is covered with a layer of fresh cancellous bone which gradually fills up the whole of the cavity (fig. 845, *b*, *c*, *d*).

(3) The compact layer forming the outside of the bone gradually spreads over this cancellous layer and eventually forms a complete cover (fig. 845, *e* and *f*).

Where the bone around the teeth has been chronically inflamed, complete regeneration of the compact tissue does not always occur, and the surface of the bone remains of an irregular character.

The alveolar process is often splintered when extraction is difficult, and the sharp edges of the bone, which are often very painful, retard healing. It is advisable to remove these projections of bone with a pair of 'nibbling' forceps, and for this purpose the instrument shown in fig. 846 is useful.

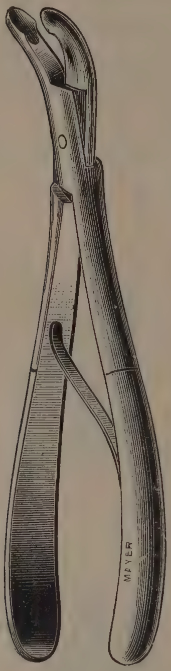

Fig. 846. (These forceps are known as Jansen's Mastoid Gouge Forceps. For the use of this block we are indebted to Messrs. Mayer and Phelps.)

In the case of molar teeth where the gum has receded the septum of bone between the roots may, after extraction of the teeth, project above the general level of the wound. The removal of the projecting bone will often expedite the healing of the wound.

(8) Extraction during the Menstrual Period.

The extraction of teeth during menstruation should be avoided unless it becomes imperative in consequence of acute suppuration. (See also p. 884.)

(9) Extraction of the Deciduous Teeth.

Although the methods employed in the extraction of both deciduous and permanent teeth are alike in actual details, there are one or two points connected with the extraction of the deciduous teeth to which attention may with advantage be directed. The degree of force necessary for the proper application of forceps to the deciduous teeth is usually less than that needed for the permanent teeth, and must accordingly be judged in each case by the sense of touch if undue damage is to be avoided; particularly is this so as the time approaches when a deciduous tooth should normally be shed; similarly, as this period approaches and the roots of the deciduous teeth become absorbed, less force will be required to displace them. The average child is a very good patient, but it must never be deceived or taken unawares; if it is told that a certain dental operation will cause pain, and the probable extent of it, it will do its best to assist, but if it has been assured that it will have no pain and suddenly finds it to be untrue, not only does the dental surgeon deservedly lose its confidence—and usually for all time—but the child's view of dental surgeons may be so altered that it may never again throughout its life go willingly to consult one. The spoiled child is anathema; apart from that it is to be regretted that ignorant parents are more apt to try to deceive and frighten children with regard to dental pain than are dental surgeons, whether it concerns the extraction of teeth or otherwise. The confidence and consent of children can generally be gained by the exercise of moral suasion after a little experience and practice.

(B) EXTRACTION OF INDIVIDUAL TEETH AND ROOTS.

(1) Maxillary Permanent Teeth.

For the removal of teeth in the maxilla the patient should be so placed as to allow the left arm of the operator to embrace the head of the patient should this be necessary. The operator should stand at the right side of the patient, and slightly in front, the first finger and thumb being placed on either side of the alveolar process (fig. 847). If the patient becomes restless, the arm may be moved so as to encircle the head and hold it firmly.

(*a*) *Maxillary First Incisors.*—The roots of the first incisors are usually cone-shaped, the anterior surface forming the arc of a larger circle than the posterior surface. Forceps for the removal of these teeth ought therefore to have the blades made to correspond to the surfaces. In removing maxillary incisors the posterior blade is applied first, care being taken to see that the edge of the instrument passes between the gum and the tooth. To dislodge these teeth a firm inward movement should be made in a direction towards the palate, this movement being followed by one in an outward direction. If this fails to dislodge the tooth from its attachments, a firm rotary motion, first to the right and then to the left, may be tried (the amount of rotation necessary being only about an eighth of the circle represented by the circumference of the root).

It is usually recommended that, in the first instance, rotation should be tried for the extraction of these teeth, but the teeth yield more readily and with less laceration of the soft tissues if the inward movement is adopted.

The extraction of the roots of these teeth does not, as a rule, present much difficulty. When the roots are moderately sound the instrument shown in fig. 832 may be used, but where the root is much decayed and lies well below the gum margin, a rather finer pair will be found more serviceable. The manner of removal is similar to that adopted when the crown is standing.

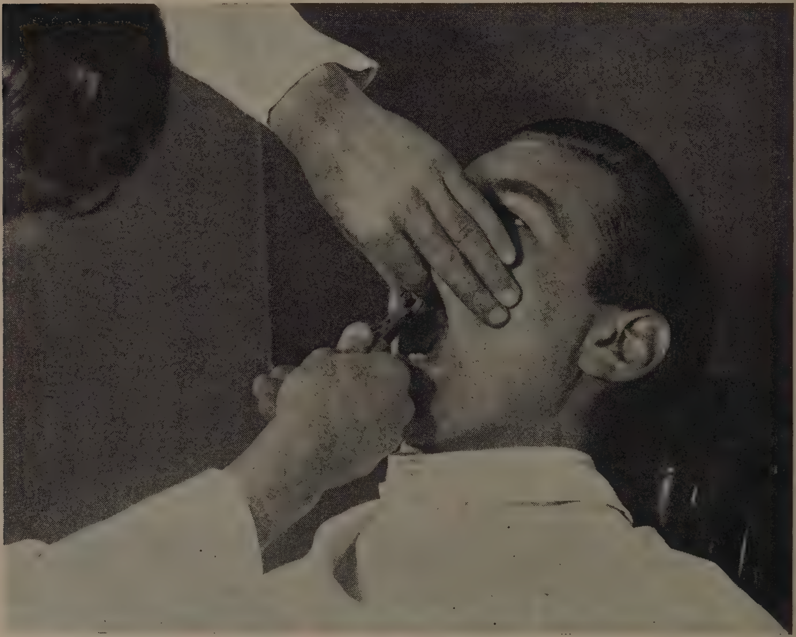

Fig. 847.

(*b*) *Canines and Second Incisors.*—The canines and second incisors are single rooted, like the first incisors; but the difference between the curves of their anterior and posterior surfaces is greater than it is in the case of the first incisors. The roots of the canines are much longer and more firmly implanted than the roots of the incisors, and more force is therefore necessary for their removal.

Forceps similar in pattern to those employed for first incisors may be used, the severance of the tooth from its attachments being brought about by force applied in an inward, followed by an outward, direction. The root of the canine being more or less three-sided, rotation cannot so well be adopted. The root of a canine tooth should be removed in the same manner as the whole tooth.

(*c*) *Premolars.—The first premolar* has usually one root flattened and more or less longitudinally grooved on its mesial and distal surfaces. If this grooving is very pronounced, the result is that the root is more or less divided into two slender terminations. The existence of such bifurcation can seldom be determined before operation, and would not alter the method of extraction, but the possibility of encountering this variation should be borne in mind and the lateral movement should be made very cautiously. The internal and external surfaces of the root are, for all practical purposes, equal in curvature.

The second premolar has usually only one root, which, in comparison with the root of the first premolar, is not so flattened in the antero-posterior diameter nor so grooved and bifurcated.

The blades of forceps for the premolars should be equal segments of the same circle; they should also be bent at such an angle with the handles as to allow the handles to clear the lower lip. The forceps shown in figs. 833 and 850 are useful patterns. In cases where patients can open the mouth widely enough to give easy access, straight forceps as in fig. 832 may be used, but often the use of these involves risk of pinching the lower lip between the handles as the blades are closed. In removing a maxillary premolar, the inner blade of the forceps should be applied first. For severing the tooth from its attachments, a slight inward movement should first be made, followed by an outward. If the socket does not then yield, the inward movement may again be made and repeated if necessary. The removal of the tooth from its socket is to be carried out by force applied in a downward and outward direction. The removal of premolar roots is carried out in a similar manner.

For the removal of unerupted premolars an anæsthetic should always be given, as the removal of the premolar is frequently difficult. Nitrous oxide is quite sufficient if the operation is confined to one side of the mouth; but a longer period of anæsthesia must be obtained if it is necessary to remove both premolars at one sitting. The most suitable instrument for the removal of the teeth is a pair of upper root forceps with rather long blades. The deciduous molar if present is first removed. In attempting the removal of the premolar, the inner blade of the forceps must be kept well inwards, the blades being thrust well upwards and extractive force applied in an inward direction. The occluding surface of the erupting premolar is directed slightly inwards, and, unless the precaution suggested is taken, the inner blade of the forceps impinges on the occluding surface and does not grip the inner aspect. The after-treatment of the wound consists in the use of an antiseptic mouth-wash.

(*d*) *Molars.—The first molar* has three roots, one palatine and two buccal. The palatine is the largest, subcylindrical in form, and often curved. The two buccal roots are placed one in an anterior and the other in a posterior position, the latter being in a plane internal to the anterior root; both are somewhat flattened; the anterior is the larger. The roots of the *second molar* are similar in shape to the first, but are usually smaller and frequently two of them are conjoined.

The third molar, when normal, has three roots, but very frequently these are all joined together and form an abrupt tapering cone, the point of which is often curved. Owing to the disposition of the roots, different forceps will be required for the removal of maxillary molars on the right and left sides (fig. 848). Of the blades, the outer or buccal should possess two grooves, the anterior being the broader and placed in a more external plane. This blade should also have a slight projection between the grooved surface for adaptation to the space between the buccal roots. The inner or palatal blade should possess only one groove. A well-made pair of upper molar forceps fits the neck of a maxillary permanent first molar perfectly. The blades should be bent at an angle with the handles, so that, when in use, the latter are clear of the lower lip. The palatine blade should be applied first, and in bringing the outer blade into place the point should be kept over the groove on the buccal side of the tooth, as this groove is a guide to the space between the outer roots. In order to sever these teeth from their attachments, force must be applied, first slightly inwards and then outwards, the movement being repeated if necessary, the removal of the tooth from the socket being carried out by exerting force in a downward and outward direction. *Too much outward movement leads to undue bending or fracture of the external alveolar plate.*

In removing the third molars the correct application of the forceps is of the utmost importance, as there is danger of including some of the soft tissue between the blades and the tooth and so causing a painful laceration. Force applied inwards, then outwards, is generally sufficient to loosen these teeth, their removal being carried out by a downward and outward movement.

For the removal of the third molars, most operators use a bayonet forcep (fig. 849) with the blades bent at a greater angle than in those used for the removal of premolars. *There is an abnormality of the maxillary molars* which may with advantage be mentioned here. It consists in the posterior buccal root being situated in a plane much internal to the anterior root—in other words, it is an exaggeration of the normal arrangement. Such teeth are termed '*oblique-rooted.*' This abnormality is met with most frequently in the third molar, sometimes in the second, rarely in the first. The difficulty encountered in extracting these teeth is that the outer blade of the forceps is apt to slip round. Oblique-rooted teeth can at times be diagnosed by noting an undue prominence of the alveolar process over the anterior

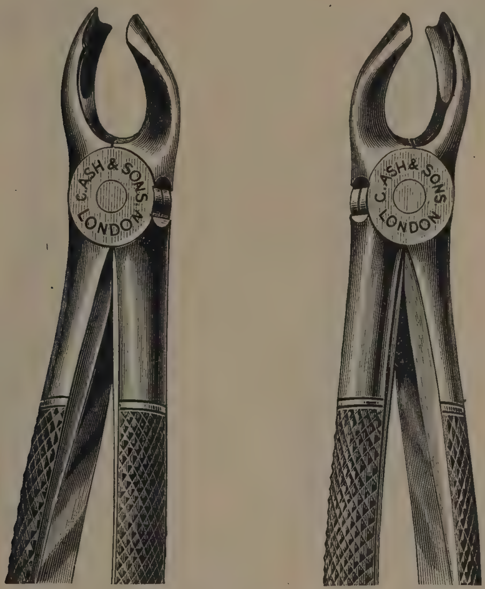

Fig. 848.

buccal root; such teeth are best removed with forceps similar to that shown in fig. 849.

Where a portion of the crown remains and caries has extended well below the gum on either the palatal or buccal side, ordinary molar forceps should be discarded and root forceps employed; useful patterns are shown in figs. 833 and 850. The removal of teeth in this condition is carried out as follows: It will be supposed that the decay extends deeply on the palatine side. One blade of the forceps should first be applied to the buccal side of the tooth and to that root which is considered to be the stronger; the inner blade should then be applied to the palatine root, care being taken to insinuate it between the alveolus and the root. The forceps should be pushed well upwards until a firm hold of the root is obtained. A firm inward movement should then be made, as this will allow the inner blade to pass still higher up the palatine root and ensure steadiness should the blades tend to ride upon the surface of the root. An outward movement should next be made, but far less than that used in extracting molars with the whole of the crown standing. This inward and outward

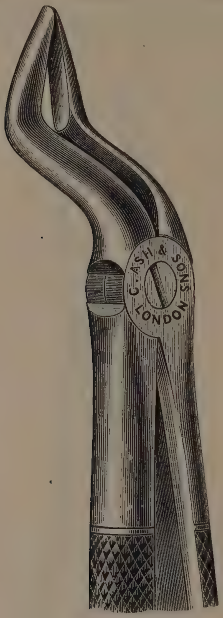

Fig. 849.

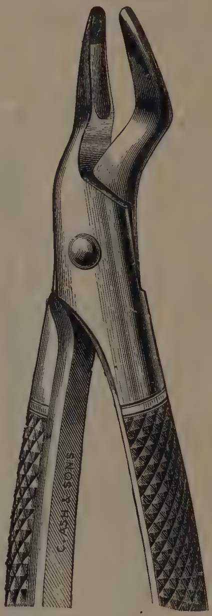

Fig. 850.

movement is to be repeated until the tooth is free, the force being principally applied in the inward direction.

When the more extensive decay is on the buccal side the order of procedure is slightly different. The first blade to be applied should be the palatine, the outer blade being closed upon whichever of the buccal roots is considered the stronger. The extractive force should be applied first outwards and then inwards, these movements being repeated if necessary, the principal force being outwards, as the object in view is to prevent the instrument from slipping off the more decayed side.

When a molar is so carious that very little of the crown remains, but all the roots are still united, root forceps should be used. The inner blade should be applied to the palatine root first, the outer blade being closed upon the stronger of the buccal roots. Inward, followed by outward, movement should be employed, the point to bear in mind being to use force towards the side of the tooth which is considered to be the weaker. Usually the three roots come away together, but if one or two roots remain they can be removed later without much difficulty.

Where the resistance presented by dense alveolar bone is very great and an unsuccessful attempt at removal has been made with ordinary root forceps, an instrument with a buccal blade similar to that shown in fig. 851 may be used. The inner blade is applied first, the outer being inserted, if possible, into the space between the buccal roots. A firm hold of the roots having been gained, extraction should be attempted by force applied in an inward and outward direction; this failing, sufficient pressure should be put upon the handles to split the roots asunder. The sharp outer blade of the forceps will then pass between the buccal roots on to the palatine root, which can thus readily be brought away. A pair of ordinary upper root forceps should be employed to remove the buccal roots.

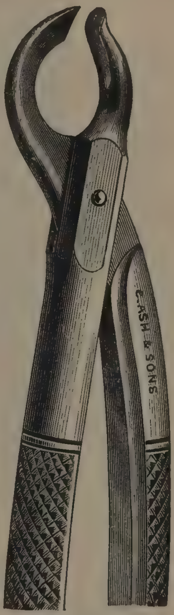

FIG. 851.

If all three roots of a molar are separate, a slight rotary movement will usually suffice for their removal.

Where there is danger of a molar fracturing, root forceps should be used in preference to ordinary forceps.

(2) Mandibular Permanent Teeth.

For the removal of mandibular teeth the patient should be placed on a low level, the head being kept a little forward and the chin depressed. The position of the operator will depend upon the tooth to be removed and the instrument to be used. When hawk's bill pattern forceps or elevators are used for teeth on the right side, the operator should stand behind and to the right of the patient, the left arm being brought round the patient's head. The thumb of the left hand should be placed on the inside and the first finger on the outer side of the alveolar process of the tooth to be removed, and the three remaining fingers under and supporting the chin. In placing the fingers in the mouth, care should be taken to depress the wrist so as not to impede the entrance of light (fig. 852).

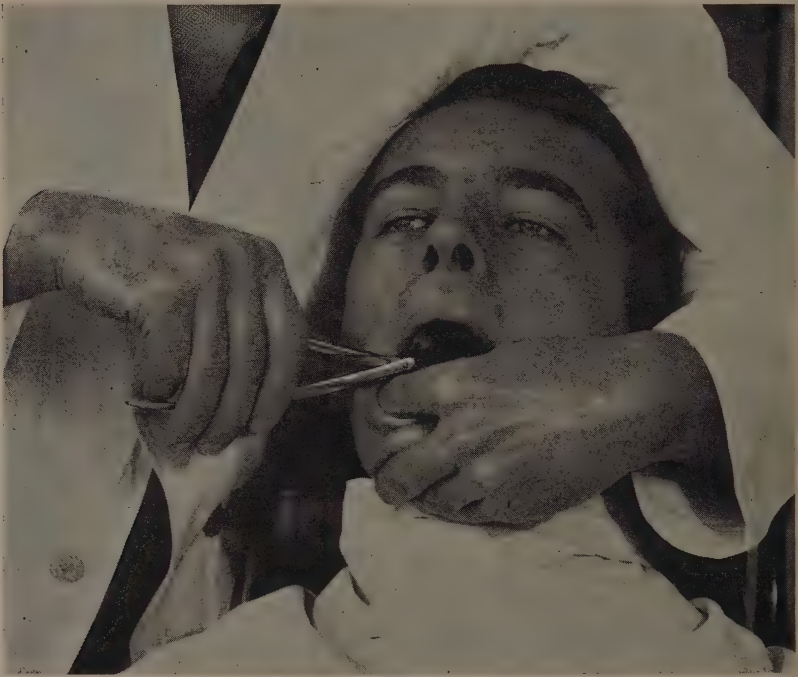

Fig. 852.

When removing the anterior teeth or those on the left side of the mouth, the operator should stand on the right side and slightly in front of the patient. The left hand should be placed as follows: the second finger on the lingual side, and the first on the labial side of the alveolar process of the tooth to be extracted, the thumb being placed under the chin (fig. 853). When employing forceps of the pattern shown in fig. 837, the operator should stand as shown in fig. 853, but it will be found difficult to place the fingers of the left hand on either side of the alveolus; indeed, they can only be used with advantage for retracting the cheek and supporting the mandible.

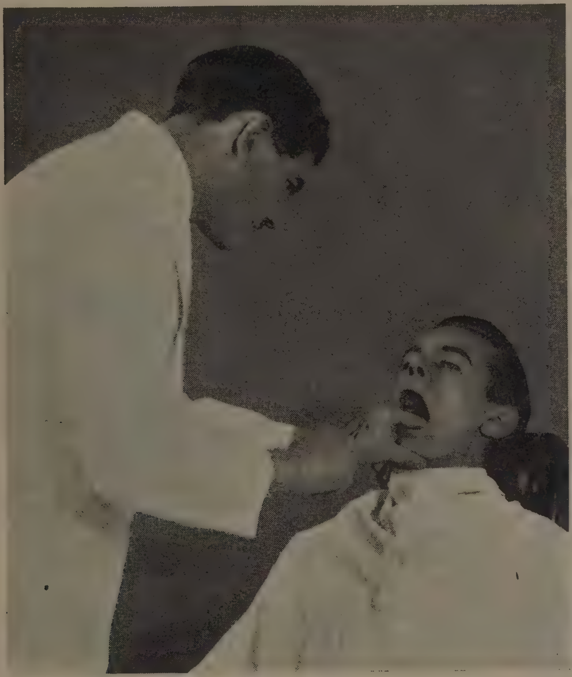

Fig 853.

In removing teeth from the mandible, the operator should be careful to guard against the sudden separation of the tooth from its attachments when raising the tooth from its socket, as, unless the instrument is well under control, damage may be caused to the maxillary teeth.

(*a*) *Incisors.*—These teeth have each a single root, which is much flattened laterally. For their removal, forceps similar to those shown in fig. 835 should be used, the blades being equal segments of the same circle. The lingual blade should be applied first, the loosening movement being made by taking the tooth slightly inwards and then outwards, the final extractive force being upwards and outwards.

The removal of mandibular incisor roots is carried out in a similar manner.

(*b*) *Canines.*—The mandibular canines have normally one root, which is flattened laterally. It is stronger and longer than the incisor roots. The removal of a mandibular canine is carried out in a manner similar to that employed for the removal of a mandibular incisor, but as the canines present more resistance than the incisors greater force is usually required.

(*c*) *Premolars.*—The mandibular premolars have normally one root, which is conical in shape. The conical shape of the root is not so marked in the first premolar as in the second, the outer aspect forming the arc of rather a larger circle than the inner aspect. Forceps similar to those shown in fig. 835 may be used, the blades being practically equal in size and shape. The lingual blade of the instrument should be applied first, the tooth being severed from its attachments by a slight rotary movement around the long axis of the tooth, first in one direction, then in the other ; should this not suffice, a slight inward, followed by an outward, movement may be tried, the tooth being raised from its socket by force applied in an upward and slightly outward direction.

The roots of mandibular premolars should be removed in a manner similar to that required for the extraction of the whole tooth. When the root lies much below the level of the gum, the extraction is often troublesome owing to the difficulty in gaining a hold with the blades of the forceps ; in such cases, if an attempt with forceps has failed, the straight elevator may be employed.

Owing to their conical shape, the pressure exerted by forceps in grasping these teeth firmly sometimes occasions their suddenly jumping with some force from the forceps after the fibres of the periodontal membrane have been ruptured ; the sharpness of the blades is the best preventive of this, but in any case the direction of a possible jump must be carefully guarded so that the tooth may not disappear at the back of the mouth.

(*d*) *Molars.*—Mandibular molars have two roots, placed anteriorly and posteriorly. The roots are much flattened, and have a tendency to curve backwards, the curving being well marked in the second and still more so in the third molar; a fusion of the two roots is at times met with in the second and, frequently, in the third molar. A section of a mandibular molar at the neck shows both the buccal and lingual surfaces to be composed of two segments of a circle touching each other at one extremity, the anterior segment being slightly the larger (fig. 854). Each blade of the forceps used for these teeth should possess two grooves separated by a projection which fits into the division between the anterior and posterior roots; for all practical purposes the blades may be made of the same size, so that one instrument will suffice for both sides of the jaw. The instrument best adapted for the removal of these teeth is shown in fig. 855. The advantages may be briefly summed up as follows: (1) A clear view of the tooth and its surroundings can be obtained during the whole period of removal; (2) force can be applied with advantage; (3) the alveolar process can be easily embraced by the fingers, or by the finger and thumb of the left hand.

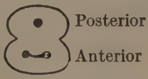

Fig. 854.

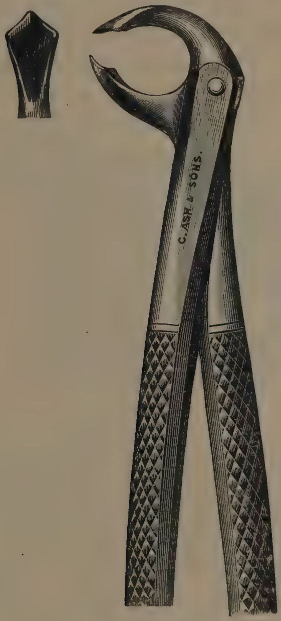

Fig. 855.

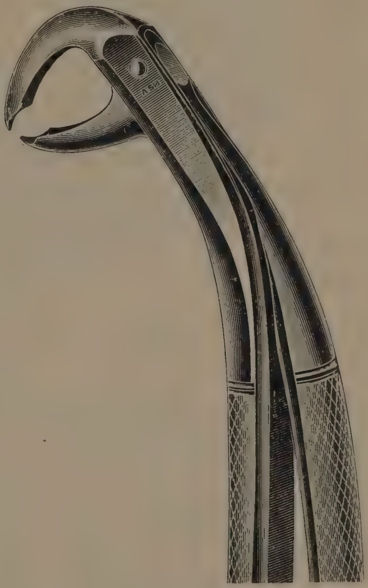

Fig. 856.

One disadvantage of this shape is the difficulty of employing much inward movement particularly for teeth inclined inwards, the extent of inward movement being limited by the proximity of the maxillary teeth; greater freedom of movement may, however, be obtained by using a similarly beaked forceps but with the handles bent at an angle as in fig. 856.

In removing mandibular molars with forceps, the inner blade should be applied first and then the outer, care being taken to get the point of the blades between the interspaces of the roots. For severing these teeth from their attachments, a slight inward movement should be made first, followed by a movement well outwards, these inward and outward movements being repeated if necessary. The removal of the tooth from its socket is carried out by force used in an upward and outward direction. The upward force exerted upon mandibular teeth should always be well under control, as not infrequently the resistance is overcome very suddenly, and without such precaution there is danger of forcibly striking the maxillary teeth. As previously pointed out, the roots of these teeth are at times curved a little backwards, and it is often needful, in removing the teeth from their sockets, to twist the forceps in a curved direction backwards.

In the *removal of the second molar*, too much outward movement is to be avoided, as the outer alveolar process is often very dense.

The third molar is best removed with a straight elevator. A glance at the illustration of this tooth (fig. 857) will show that the roots have a well-marked curve backwards, in addition to which the bone forming the socket of this tooth is stronger than is the case with the anterior molars. The removal of the third molar has therefore to be accomplished by using force in a direction upwards and backwards; in other words, in the curve following the arc of the circle formed by the roots. This movement cannot well be carried out with forceps, but is easily accomplished with the elevator as follows (it being assumed that the second molar is in place): Hold the elevator as shown in fig. 843, and insert the blade between the anterior surface of the root and the alveolar process, keeping the flattened side of the instrument as far as possible parallel with the root surface. Then force the blade downwards towards the apex of the root and rotate the handle away from the direction in which the tooth is to be moved. This has the effect of raising the tooth in its socket and at the same time displacing it backwards. (The edge of the elevator which is to be brought into contact with the surface of the root should be sharp so as to cut somewhat into the cement.) If this movement is not effective the handle should again be raised and the flattened end of the instrument brought parallel with the anterior surface of the root, the extractive movement being repeated until the tooth is completely raised from its socket. In using the elevator, special care must be taken to protect the tongue with the thumb or the fingers of the left hand, so as to provide against the instrument slipping and penetrating the tongue.

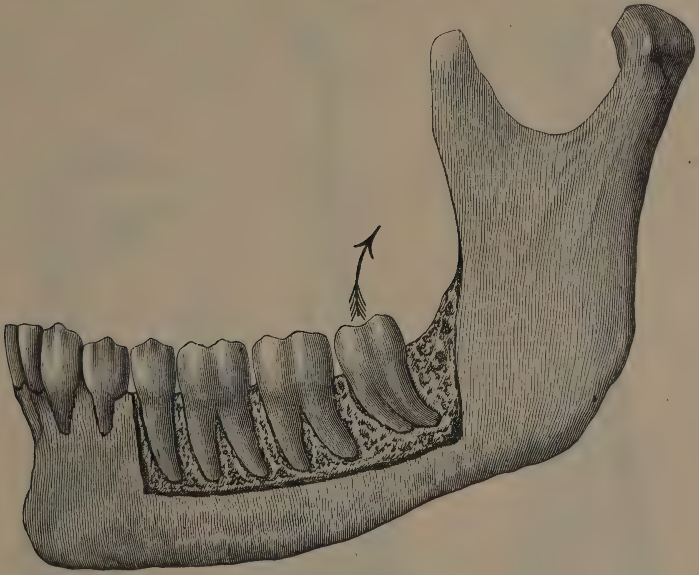

FIG. 857.

With the third molar there is a tendency for the gum to adhere tenaciously to the posterior aspect of the neck of the tooth. When this happens it is better simply to raise the tooth from its socket with the elevator or forceps, as the case may be, and then separate it with a curved pair of scissors. By this method a severe laceration of the gum may at times be avoided.

The removal of mandibular molars when a portion of the crown is standing, but the caries has progressed below the gum on either the buccal or lingual aspect, is carried out with root forceps of shape shown in fig. 835. A similar condition in maxillary molars and the method of their removal was referred to on p. 834. The principles there enunciated apply to the removal of the mandibular molars. The main points to bear in mind are to apply the blades of the forceps to the stronger root, and to use the principal force in the direction of the weaker wall.

Where the roots of molars are still united, root forceps should be used, the blade being first applied to the lingual surface of the stronger root. A firm hold having been obtained, the root may be removed by employing force in the same way as with ordinary molar forceps. Both roots will usually come away together. If only one root is extracted, the remaining root can easily be removed with the same forceps or with a curved elevator (figs. 858 and 859). The curved elevator should be insinuated between the root and the alveolar process so as to force the tooth into the empty socket, or the elevator may be placed in the empty

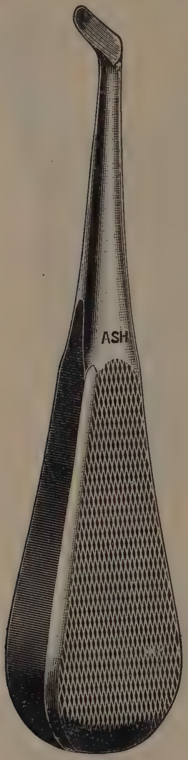

Fig. 858.

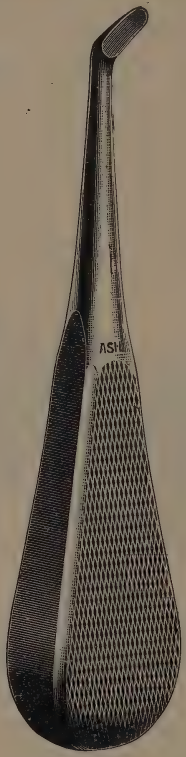

Fig. 859.

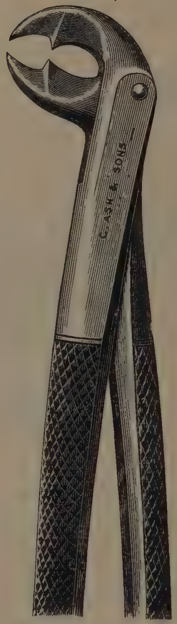

Fig. 860.

socket and rotated; this will force the point through the septum of bone, when it will engage on the root near its apex and raise it up.

With roots of mandibular molars when the bone presents great resistance, forceps with cutting blades may be used (fig. 860). The blades are inserted on the lingual and buccal aspects of the roots in such a manner that the points pass into the space between the roots. The handles are then closed and an attempt is made to remove the roots in the ordinary way, but, should this prove unsuccessful, the handles must be forcibly closed so as to divide the roots, which can then, as a rule, be removed with ordinary root forceps.

The advantage of splitting roots in a case similar to that shown in fig. 861 is apparent, as each root can then be removed in the line of its inclination.

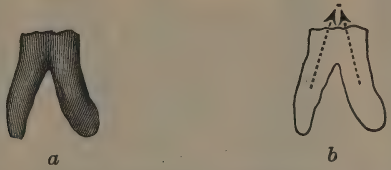

Fig. 861.—(*a*) Mandibular molar with divergent roots; (*b*) the dotted lines show the direction in which the roots can be removed if the tooth is divided as suggested in the text.

Where the roots are separated their removal is carried out with root forceps, an inward and outward movement being usually sufficient. The roots of third molars may be removed with a straight elevator when the second molar is standing. The *modus operandi* is similar to that used in extracting the whole tooth.

At times the mandibular molar teeth are tilted so that the crown surfaces stand towards the tongue. Their removal is best carried out with instruments of the pattern shown in figs. 837 or 856, as the handles of forceps of the hawk's bill pattern would come in contact with the maxillary teeth and impede the inward movement which is so necessary for the removal of teeth in this position.

(3) Deciduous Teeth.

For the removal of maxillary incisors and canines, a small pair of straight forceps of the pattern shown in fig. 862 should be used.

The deciduous first molars are best removed with a pair of forceps like fig. 863.

The mandibular incisors and canines require a small pair of hawk's bill forceps similar to the shape shown in fig. 835. For the deciduous molars a small pair of forceps similar to that illustrated in fig. 855 should be used.

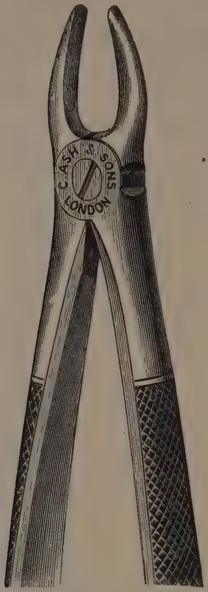

Fig. 862.

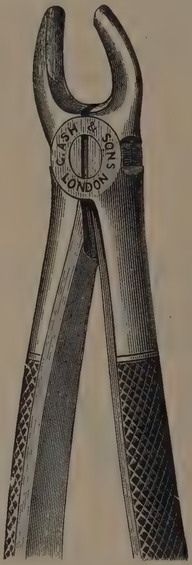

Fig. 863.

In removing deciduous teeth, care must be taken not to drive the forceps too far in or the permanent teeth may be injured, and this danger should be specially guarded against in the case of the deciduous molars, as the roots of these teeth practically embrace the crowns of the premolars.

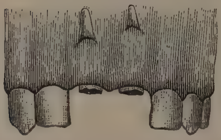

Fig. 864. (From 'Dental Surgery and Pathology.' A. Coleman.)

Roots in the condition shown in fig. 864 are best removed with an elevator as follows: The thumb of the right hand being placed on an adjacent tooth so as to gain a hold, the point of the elevator is placed beyond the end of the root and pressure applied. In a few cases it may be necessary to incise the gum before using the elevator. For the small pieces of the deciduous teeth which persist and become wedged in between the permanent teeth, small curved elevators will be found most useful.

(C) THE EXTRACTION OF MISPLACED TEETH.

The extraction of a misplaced tooth is perhaps the greatest test of the skill of an operator, and although it is impossible to give a complete list of the various malpositions met with, those most commonly seen will be mentioned and the usual method of removing such teeth indicated.

(1) Maxillary Teeth.

(*a*) *First Incisors.*—The extraction of an irregularly placed incisor such as the right first incisor shown in fig. 865 is best carried out with an instrument similar to that shown in fig. 866, the fine inner blade being applied on the palatal side and the broad blade on the labial. Extractive force should be applied principally in the outward direction, and, if this is not sufficient, slight rotary movement should be tried. Where there is less room between the approximal teeth, the projecting tooth may be removed with a pair of straight forceps (fig. 832), the blades being applied to the medial and distal aspects of the root. The blades should not be driven very far up, and the loosening of the tooth should be accomplished by a slight rotary motion, but care must be taken to avoid loosening the approximal teeth.

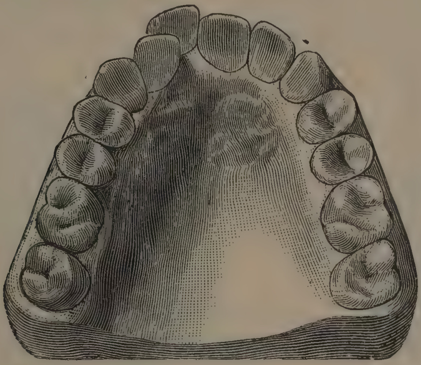

FIG. 865.

(*b*) *Second incisors* lying internal to the arch (see fig. 867) can be removed with the forceps shown in fig. 866, by placing the fine blade on the labial and the broad blade on the palatal side of the tooth. Extractive movement should be made inwards, followed by very slight outward movement ; this failing, rotation should be tried, but, as pointed out on a previous page, this form of movement is not so suitable for second as for first incisors.

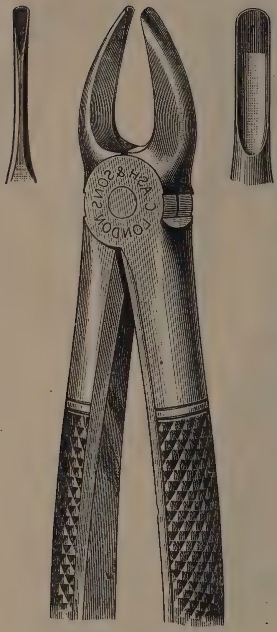

Fig. 866.

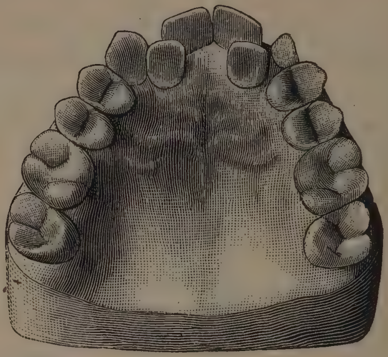

Fig. 867.

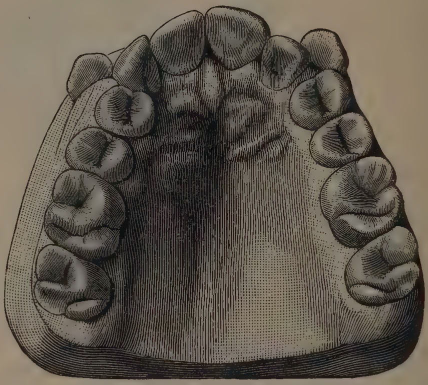

Fig. 868.

(c) *Canines* placed high in the arch, as shown in fig. 868, may be extracted with a straight pair of forceps (fig. 832), the blades being placed on the medial and labial aspects of the root. Extraction of such teeth is sometimes difficult. Slight but firm rotation may first be tried ; if this fails to loosen the tooth, slight lateral movement may be attempted, the force being applied towards, and then away from, the mid-line of the mouth.

(*d*) *Premolars* misplaced, as shown in fig. 869, can be removed with forceps similar to those depicted in fig. 833, the blades being applied on the anterior and posterior aspects of the tooth. Force should be applied in a backward and forward direction, the movements being repeated alternately until the tooth is loosened in its socket. A premolar placed as shown in fig. 870 can be removed with forceps similar in form to those depicted in fig. 833, with the outer blade strong but narrow. The extractive movement should be made mainly in an inward direction.

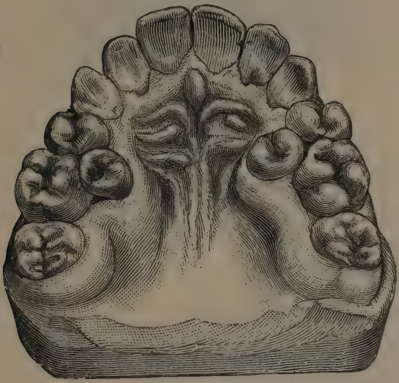
FIG. 869.

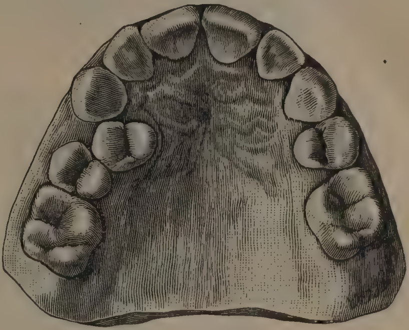
FIG. 870.

(2) Mandibular Teeth.

(*a*) *Incisors* misplaced, as shown in fig. 871, may be removed with ordinary lower root forceps (hawk's bill pattern), the blades

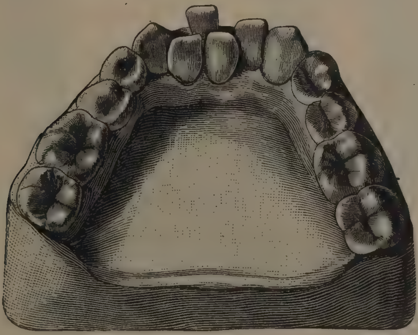
FIG. 871.

being placed on the medial and distal surfaces of the root and movement applied in a direction towards and away from the

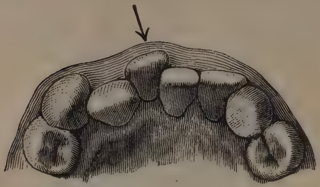

FIG. 872.

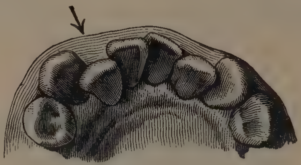

FIG. 873.

mid-line of the mouth. When the crowding is not severe (fig. 872), forceps of the hawk's bill pattern with a strong but narrow inner blade should be used (fig. 874), and the principal

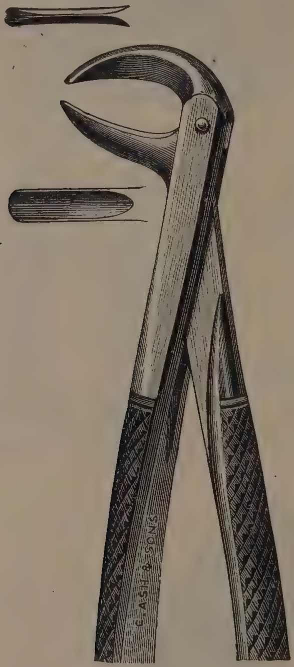

FIG. 874.

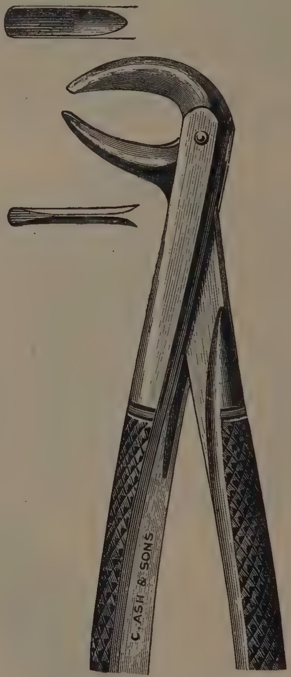

FIG. 875.

extractive movement made in an outward direction. For an incisor placed as shown in fig. 873, the narrow blade should be the outer one (fig. 875), and the principal force should be applied in an inward direction.

(*b*) *Premolars* placed as shown in fig. 876 are sometimes difficult to remove. One of the most useful instruments for their extraction is a pair of upper root forceps (Read's pattern, fig. 833), which should be held so that the concave curve of the blades is downwards. The blades should grasp the root on its anterior and posterior surfaces. Slight rotary movement may first be attempted, followed by lateral motion. These movements should be continued until the tooth is found to yield. The application of too much force may lead to a fracture which would be extremely difficult to deal with.

Where crowding is not very marked and the tooth is more in the normal line of the arch, a forceps with a narrow outer blade will suffice (fig. 875). Extractive force should be used principally towards the median line of the mouth, and this may be combined with slight rotary movement.

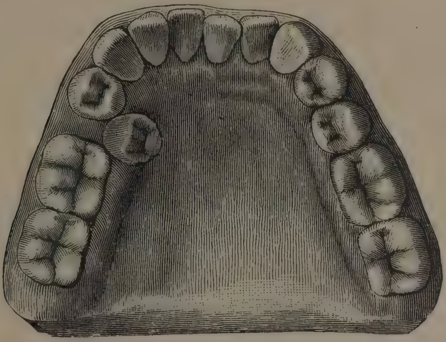

FIG. 876.

(*c*) *Third molars* when *partly erupted or misplaced* are amongst the most difficult teeth to extract. The position of the misplaced tooth may amount only to a slight tilt forwards, or the tooth may be placed horizontally, the occluding surface of the crown impinging on the posterior root of the second molar. In some instances the tooth may be so placed that the occluding surface looks downwards and inwards. An examination of specimens showing misplaced mandibular molars (figs. 113 to 116) shows: (1) That the roots of these teeth are to some extent covered by the ramus of the mandible; (2) that the root of the tooth is in close proximity to the mandibular canal; and (3) that only a small amount of bone exists at the posterior aspect of the second molar.

Wherever there is reason to expect difficulty in removing a third molar, a radiogram should be obtained in order to ascertain the relation of the tooth to its surroundings.

When the tooth is in a normal direction, but retarded in erupting through want of room in the arch, it is a good plan, before attempting removal, to remove the overlying gum in order to obtain as clear a view of the tooth as possible. Where the tooth is slightly tilted, the curved elevator will often be found a useful instrument for removal ; the blade of this instrument can usually be inserted under the crown and the tooth prised upwards. Where the tooth is firmly embedded in the bone, the muco-periosteum covering the alveolar process should be raised and the bone surrounding the tooth cut away before removal is attempted. The wound left after the removal of the tooth should be syringed regularly until healing is complete. Attempts to remove such teeth by placing a curved elevator under them and forcibly prising them upwards invariably lead to failure, and may, if too much force is used, lead to fracture of the jaw.

(3) The Extraction of Unerupted Misplaced Teeth, Impacted Teeth and Buried Apices. Alveolectomy.

The removal of unerupted misplaced or impacted teeth may be necessary for several reasons : if impacted they may in their efforts to erupt cause the absorption of part of a tooth which is in their way, and so, by involving its pulp give rise to pain of greater or less intensity and continuity. Frequently there is a narrow sinus from the surface near to the crown leading to the potential space around the crown ; infection travels along this channel to this potential space and ultimately gives rise to changes of a chronic inflammatory nature, such as productive periodontitis, rarefaction of a part of the tooth and sclerosing of adjacent bone ; these changes and the absorption of toxic material thereby brought about may give rise to pain or the effects of toxic absorption. Sometimes a tooth germ is congenitally misplaced and in the endeavour of the formed tooth to erupt signs or symptoms additional to or in place of those already mentioned are caused which make it desirable or necessary that it be removed. The teeth which are most frequently misplaced or impacted in this manner are mandibular third molars, maxillary permanent canines, mandibular second premolars, and maxillary premolars, though any tooth may on occasion be found misplaced.

It is the custom now in many of these cases to remove such teeth by what is called the **surgical removal of teeth**; the indications for such procedure will depend largely on the skill of the individual operator. Extraction by means of forceps and elevators is, of course, surgical removal, but in some cases additional surgical procedure in conjunction with the use of these is of such value in lessening the trauma and damage which might otherwise be done and in hastening healing that it has now come to be an approved method in certain circumstances.

It would be out of place here to attempt to give a detailed description of technique suitable to every case; each case must obviously be treated on its merits, but the broad principles of the procedure may be indicated. Whenever any such operation is contemplated radiograms from several aspects, preferably stereoscopic, are a necessity.

The principles involved are that a curved incision is made through the mucous membrane to one side of and concave towards the area through which it is desired to remove the tooth. The size and shape of the flap to be reflected are of considerable importance and must be so arranged that the base of the flap is towards its blood supply; in the case of the maxillary canine this is derived from the descending palatine branch of the internal maxillary artery; passing through the posterior palatine canal this vessel runs forward in a groove midway between the mid-line and the edge of the gum to the incisor (Stensen's) canal, where it anastomoses with the nasopalatine artery. The mucous membrane is then raised from its attachments to the underlying bone, and the flap folded back and held in this position by retractors; the bone is then cut away over the tooth by means of burs on the dental engine or by chisels, used either by hand or with a mallet as its density may indicate or, if the bone is very thin, it may be cut through and reflected, still attached to the mucous membrane, though this is not often possible.

When the tooth has been freely exposed it is freed from its attachments, by forceps or elevators or such other levering instrument as may be convenient, and removed; after hæmorrhage has been controlled the wound is washed with warm saline and the reflected flap of mucous membrane replaced and stitched into position and the wound allowed to heal; if it is infected it may be necessary to leave one corner (the most dependent) open and insert a gauze drain; this opening may be used for lavage later on should it be necessary. Such technique, the position and direction of the incision being decided by the area to be opened, is suitable for the removal of such buried teeth as misplaced maxillary canines, which are generally removed from the palatal aspect (see figs. 877 and 878), and impacted mandibular second premolars, which are removed from the buccal aspect. An example of this latter is shown in fig. 879; here the mandibular second premolar is seen impacted between the first premolar and the permanent first molar. In the case of an impacted tooth, in order not to injure teeth against which it may be impacted after it has been exposed, it is sometimes advisable to cut it in two with a bur, or having notched it with a bur, to divide it by a blow with a mallet and chisel; this also facilitates its removal, but great care is necessary in the latter case lest more damage than is desirable be done.

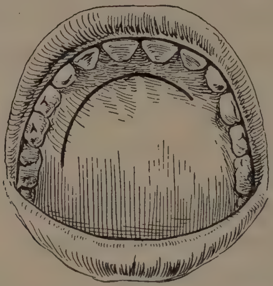

Fig. 877.—Incision for unerupted canine. (From the *Dental Record.*)

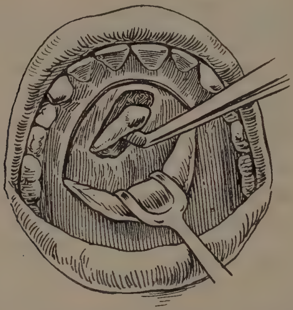

Fig. 878.—Removal of an unerupted canine. (From the *Dental Record.*)

In the case of **misplaced or impacted mandibular third molars** the same principles are involved, the bone in this region is usually denser and more difficult to remove and the gum overlying the tooth is frequently removed instead of being reflected and replaced. A series of bone chisels, gouges, picks, and elevators (figs. 880 to 883) has been devised specially for the surgical removal of this tooth by G. B. Winter, and in his hands their successful use is amply demonstrated, but it must be remembered that it is the skill of the individual and not the instrument used which results in the successful removal of these sometimes very difficult teeth.

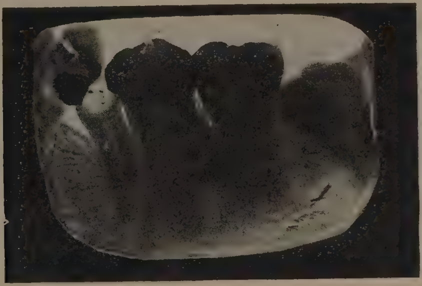

FIG. 879.

In the removal of these teeth it is usual, if bone is to be removed, to remove it from the buccal rather than the lingual aspect, though it is considerably thicker on the buccal aspect in this region. Bearing this in mind, and the surgical ideals of drainage from the lowest point and diminution of the area to be drained to a minimum, a procedure has been designed by Warwick James [1] for their removal from the lingual aspect ; stating that accessibility was greatly increased, lighting improved and the operation more controlled, he describes the operation as follows :—

'A general anæsthetic is used. Dr. Bernard Johnson, to whom I am indebted for help, uses an intratracheal tube passed through the nose.

'The operator sits at the head of the table. The patient's head is fully retracted and fixed as firmly as possible with a head-clamp. A modified Boyle-Davis gag (without anæsthetic tube) is used to keep the mouth open and depress the tongue. A head-mirror or head-lamp is employed by the surgeon to obtain the necessary lighting of the field of operation whilst the assistant uses a head-lamp.

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), 1935, xxix, 9.

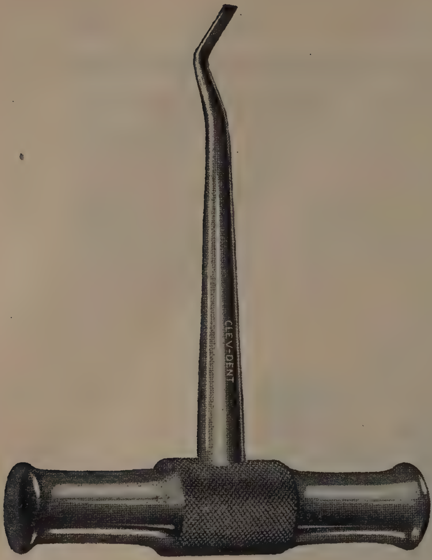

FIG. 880.

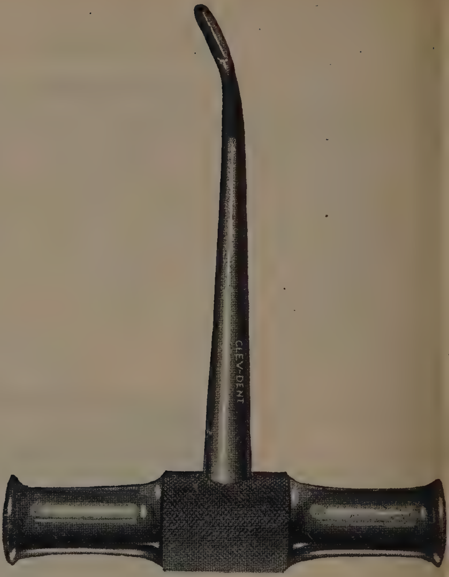

FIG. 881.

Two forms of Winter's bone cutting instruments. (The Cleveland Dental Manufacturing Co.)

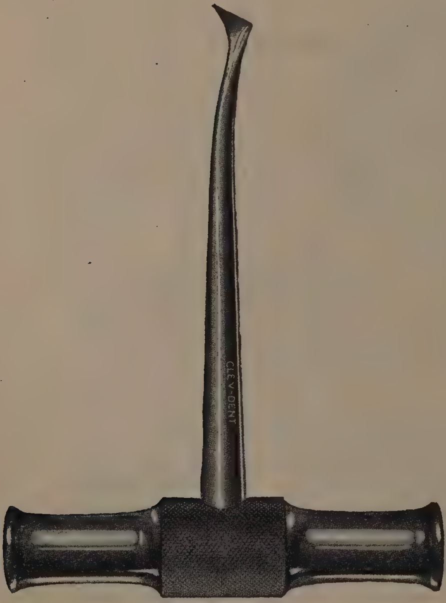

FIG. 882.

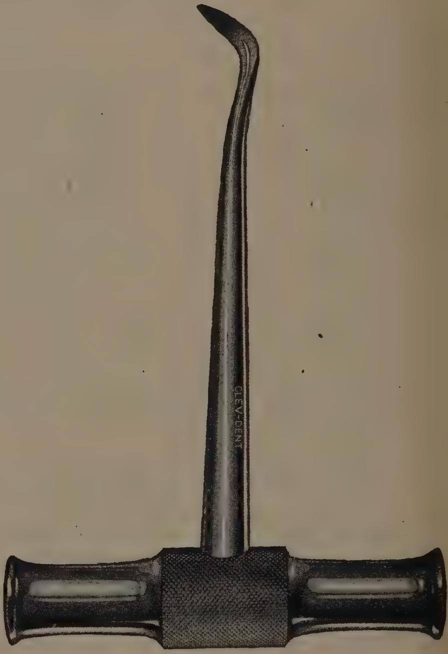

FIG. 883.

Two forms of Winter's elevators. (The Cleveland Dental Manufacturing Co.)

'The position does not demand packing of the throat, but this is advisable both in the event of hæmorrhage and in order to catch chips of bone. If the tongue bulges up at the side of the spatula on the gag, a single swab can be tucked in. The incision is made behind the second molar from a point on the lingual aspect about level with the apices of this tooth (fig. 884). It passes as a curve about ¼ in. behind the tooth, keeping more or less equidistant from it as far as the fold of the cheek. If the incision is too far back the lingual nerve may become exposed. Additional incisions will be necessary—one of about ¾ in. to 1 in. at the outer end of the first incision towards the base of the coronoid. A posterior flap is then raised. A chisel starts the separation from the bone most readily (fig. 885) and separates the mucous membrane from the bone more effectively than a periosteal elevator. When there is tension, or when further space is required, the incision is extended, possibly higher on the coronoid, or towards the cheek, or a nick is made towards the pharynx at the lingual end. It is remarkable how small a nick will permit of considerably increased retraction of the tissues.

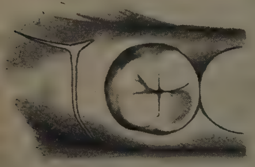

FIG. 884.

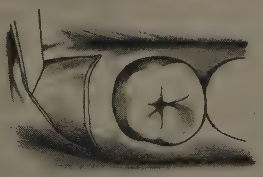

FIG. 885.

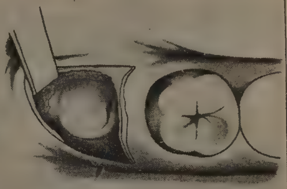

FIG. 886.

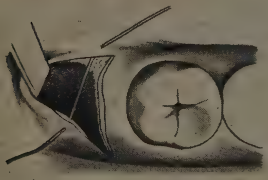

FIG. 887.

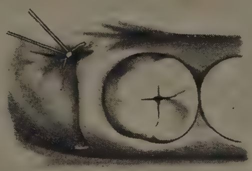

FIG. 888.

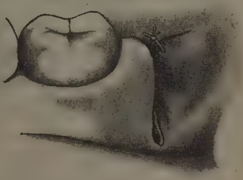

FIG. 889.

(Figs. 884 to 889 from *Proc. Roy. Soc. Med.*)

'When the tooth and the bone overlying it are freely exposed, a wedge-shaped piece of bone is removed from the inner plate. It is best to employ the chisel at a point as far posterior as it is desired to go and to make the anterior cut secondarily. The actual removal of this wedge-shaped piece will vary according to the position of the tooth. The bone over the lingual and distal aspects of the tooth must be cut away until complete exposure is obtained (fig. 886). If the tooth is situated buccally, as is often the case, part of the base of the coronoid must be removed. In all cases the bone behind the tooth must be cut away, but on no account must that in front be disturbed. The tooth is then loosened in its socket with forceps ; it is better to avoid using elevators as far as possible, although there are occasions when they are useful. Great force must be avoided, and if it appears to be demanded, more bone should be cut away.

'When operating on the right side of the mouth the ordinary hawk's-bill forceps are found to suffice but, the arm of the Boyle-Davis gag being on the left side, it is easier to use upper forceps on that side, the removal generally being effected by upper root forceps. In order to loosen the tooth upper molar splitting forceps will frequently obtain a grip upon the tooth that none other will achieve. The displacement of the tooth should be effected quite easily without any force coming against the bone at the back of the second molar, or, if possible, in any way injuring the strip of mucous membrane that has been left behind this tooth. After the tooth has been removed sharp angles of bone are rounded off. It is most important that the wound should be cleared of any loose fragments. Then a catgut suture may be inserted towards the lingual end of the posterior flap and stitched to the buccal flap at a point corresponding with the end of the first incision (fig. 887).[1] It should be so arranged that, when tied, a definite space is left from which drainage can take place at the lowest point (figs. 888 and 889). There should be no puckering of the flap, nor should there be tension. The flap itself should come in contact with the mucous membrane at the back of the second molar. When this has been done a swab should be taken and gentle pressure made upon the flap so that it is brought as firmly as possible into contact with the tissues beneath. Finally, a long strip of gauze placed in the cheek is packed firmly on to the top of the wound and the mandible closed against the maxilla, a short end of the gauze hanging out of the mouth.

'I have not described any cases in detail, but a sufficient number of operations have been carried out to enable me to express considerable confidence in the procedure. Deeply-situated teeth have been removed, when formerly extraction of the second molar would have been necessary in order to gain access. In nearly all cases the freedom from after-pain, the slight amount of swelling, and the almost complete absence of trismus have been most encouraging. My chief error at first was due to an endeavour to close the wound too firmly. Should a stitch be necessary to hold the soft parts in position, it should be removed early if swelling or pain is marked, even on the day following the operation.

'In giving details of the operation it has been assumed that the tooth is deeply situated. As is well known, many variations in position occur and quite commonly the tooth is so closely related to the second molar that the mucous membrane behind this tooth cannot be kept intact.'

Owing to the frequency with which the mandibular third molar is misplaced or displaced, and the difficulty that may be encountered in its removal under these circumstances in adult life, C. Bowdler Henry has devised an operation for the removal of this tooth in its developmental stage, either before or soon after its calcification has commenced, and when it is more superficially placed than later on. He states that probable impaction may be diagnosed from radiograms between the ages of nine and eleven years, and summarises the indications for its prophylactic enucleation at this stage as follows :—

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[1] The stitch may often be placed more posteriorly in order to facilitate drainage.

(*a*) In early cases, before calcification of M_3, when the crown of M_2 is crowded against the root of M_1 and when M_3 crypt is sharply tilted behind and above M_2 and crowded upon it (compare figs. 890 *A* and *E*).
(*b*) At a later stage, after calcification of the cusps, when the orientation of the crown of M_3 is seen to be abnormal in either a mesio-oblique direction, as in fig. 890 *F*, or in a lingual direction, as in fig. 890 *D*, especially when there is further evidence also of molar crowding as shown by M_2 being pressed under the posterior convexity of M_1 (figs. 890 *B* and *F*).
(*c*) When the molar teeth, and especially M_3, are abnormally large (fig. 890 *G*).
(*d*) When M_3 (mandibular) is of normal size but M_3 (maxillary) is abnormally small or congenitally absent.
e) When M_3 is grossly deflected by a supernumerary tooth (fig. 890 *H*).
(*f*) When through the early loss of the deciduous molars, M_1 and M_2 having tilted forward, it is proposed to push these molars backward in order to allow PM_1 and PM_2 to erupt into their proper places.

He states :—

'Although it is possible to perform this operation under nasal nitrous oxide and oxygen anæsthesia or even under block novocaine with suitable premedication, these cases are most conveniently operated upon under endotracheal narcosis. Absolute asepsis is imperative, and for this reason patients cannot be suitably dealt with in a dental surgery; they should be hospitalised. Infection in a crypt may easily lead to osteomyelitis and all its sequelæ.

'With the mouth open and suitable retractors in place, the vestibulum of the cheek is stretched so as to bring the ascending ramus into prominence. To effect this, a rounded retractor (such as a Frankel tongue depressor) is pushed backward along the buccal sulcus in the mouth to the outer side of the ascending ramus. A slightly curved incision (fig. 891 *A*) is then made, beginning in front at the posterior limit of the tight pad of gum which overlies the second molar and extending for three-quarters of an inch upward. The convexity of the curve should be toward the midline of the mouth. The incision is made deeply to the bone and passes through fibres of the lower attachment of the temporal muscle, sometimes severing a nutrient vessel. The orifice of the third molar crypt is

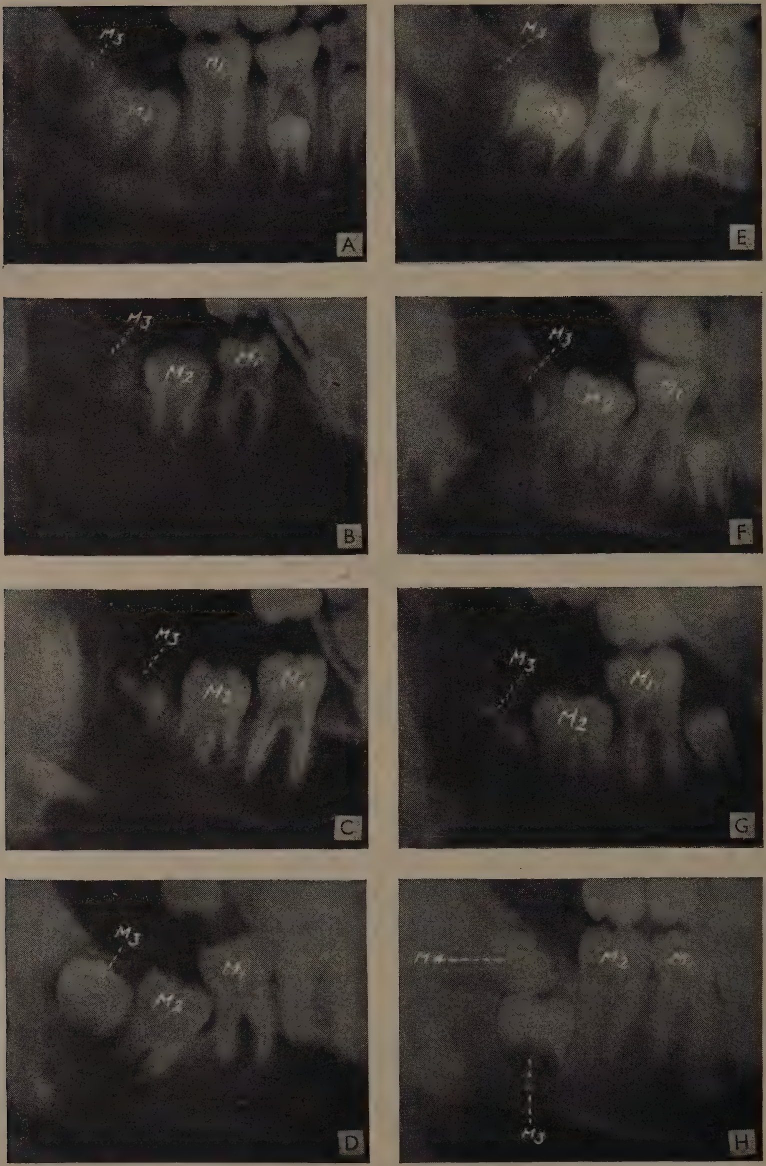
M3
M1
M2
A
M3
E
M3
M2
M1
B
M3
M2
M1
F
M3
M2
M1
C
M3
M1
M2
G
M3
M2
M1
D
M1
M2
M1
M3
H

FIG. 890.

seen at once, and the bone is bared with a narrow raspatory over an area half an inch square. The sides of the wound are held open with toothed retractors, and the roof of the crypt is removed. In young patients the roof is never complete and is as thin as eggshell, so that it can very easily be removed by hand pressure, or more neatly by light blows on a half-inch mastoid chisel with a mallet. Four strokes usually suffice to lift off a square from the roof of the crypt and give ready access. An important point to remember, however, is that rapidity of operation depends on getting the calcified mass out easily. The novice is liable to operate through too small an opening. If the cusps only are calcified, a sharp spoon, shaped like a large dental excavator, readily crushes the occlusal surface ; but if the crown is calcified, it will not break and the orifice must accordingly be made very much larger.

'The advantage of operating soon after the beginning of calcification is obvious. In the early stages (no later than that of fig. 890 *F*) the crypt is distended almost to its full size but the calcified mass is still small. There is therefore plenty of room between it and the crypt wall for inserting instruments. Later on, the calcified crown fills the whole of the crypt (as in fig. 890 *H*) and its manipulation becomes progressively more difficult.

'When the overhang of the roof has been removed as far as is necessary, a flat spoon is passed down the side of the crypt and its contents are enucleated. Usually the first object to be delivered is a square, firm, pale pink disc, the dentine papilla, which squeezes out. Further curettage either breaks up the calcified mass, which then comes away piecemeal, or delivers it whole. The

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EXPLANATION OF FIG. 890.

Radiogram of the molar regions of children ranging between seven and thirteen years, illustrating the indications for prophylactic enucleation of the M_3 follicles.

Radiogram *A* shows normal development at the stage when the crypt of M_3 is well formed but before calcification of this tooth has commenced. Radiogram *E* shows a similar stage but in a case of molar crowding.

Radiograms *B* and *F* show cases corresponding to *A* and *E* at the stage when the cusps of M_3 have just calcified. In *B* there is an abundance of room between the three molars, and the tilt of M_3 is not excessive. This case is normal. In *F* the molars are crowded together. the convexity of M_2 lying in the concavity of M_1, the tilt of M_3 is excessive and its impaction inevitable. Cases *E* and *F* are suitable for M_3 enucleation.

Radiogram *C* shows a case of normal development with M_3 of subnormal size and should be compared with radiogram *G* from a patient of the same age in which all the molars are large and crowded upon each other. In *G*, M_3 is too wide in its anteroposterior diameter to erupt, and since its impaction is inevitable, its early enucleation is indicated.

Radiogram *D* shows a case of impaction of M_2 against M_1 and the complete lingual rotation of M_3, which should be removed.

Radiogram *H* shows a case of a well-formed supplementary molar, M_4 developing above M_3 and impacting it. M_4 should be removed.

Both radiograms *D* and *H* illustrate the value of early radiographic examination. An easier operation with less trauma and a better result might have been expected had these two patients been diagnosed several years earlier.

(From *American Journal of Orthodontics and Oral Surgery*.)

connective tissue of the follicle separates easily from the bony wall and is removed. The field is kept dry with a suitable aspirator and finally, in case any calcific material and bone débris are left behind, the crypt is flushed with saline.

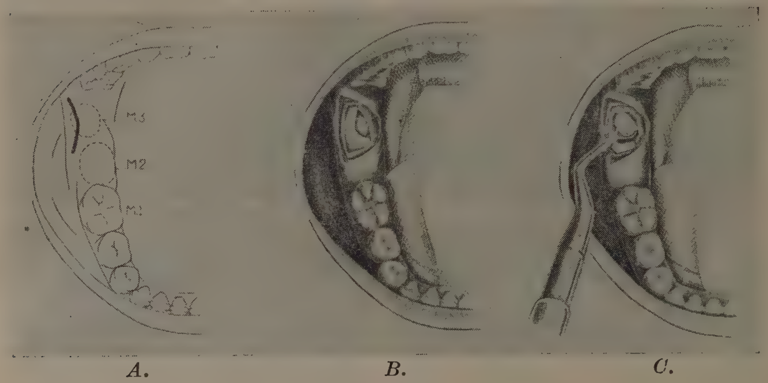

Fig. 891.

The stages of operation for the enucleation of a right mandibular third molar follicle. *A* shows the incision on the ascending ramus. *B* shows the integuments retracted and exposing the orifice in the roof of the bony crypt. *C* shows the crypt opened up and the delivery of the calcified mass. (From *American Journal of Orthodontics and Oral Surgery.*)

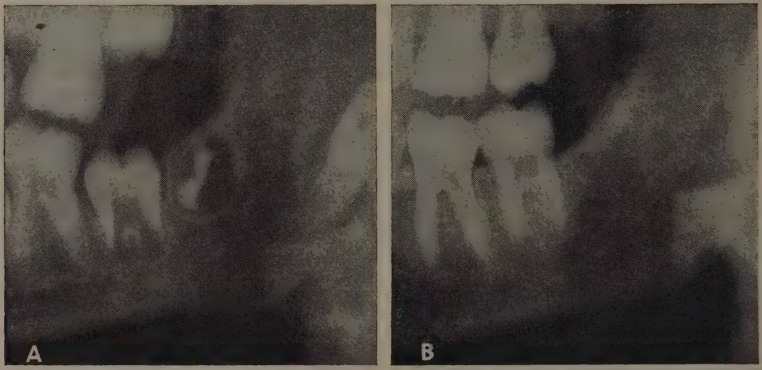

Fig. 892.

Radiograms of a case of potential impaction of the mandibular third molar, treated by enucleation of the follicle in a child aged nine and one-half years. *A*, Before operation ; *B*, twenty-two months after operation. (From *American Journal of Orthodontics and Oral Surgery.*)

'On account of occasional hæmorrhage into surrounding tissues it is best to leave the wound unsutured and to apply pads of cotton wool on the face under a tight bandage. Any seepage of blood comes out through the wound, which invariably heals by first intention. Provided the case is handled with aseptic precaution, there is no need to introduce antiseptics or plugging.

'The operation is quickly and easily performed. Although the tidying up, if scrupulously done, may take several minutes, the actual removal of the tooth germ should not exceed a minute, and the procedure contrasts most favourably with the difficulty of extracting the same tooth when the crown is formed, and still more when it is deeply buried or horizontally placed with perhaps divergent curving roots. The trauma is less and the patient benefits by losing the tooth at a time when the bone is soft and healing is rapid, and before infection has crept in. Instead of a socket which may take six weeks to heal and give trouble and postoperative pain, the incision of this operation heals by first intention and the crypt is quickly obliterated. There is no disability. The patients are allowed up on the following day.' [1] (See fig. 892.)

The principles of clean incised wounds the flaps of which may be replaced later, and the chiselling or cutting of bone by purposeful movements rather than its haphazard breaking and bruising, all tend to lessen the amount of damage done in the removal of such teeth, to lessen pain and to expedite healing. A clean view must be obtained and one's sense of touch so educated that one knows exactly how and where the tooth lies. Otherwise one is working in the dark and against unknown resistances. Small trephines, for use on the dental engine, are made for the removal of portions of bone ; these are not so much of use for gaining access for the removal of teeth as for the removal of small buried apices of teeth after their localisation by radiograms.

The operation of **alveolectomy** is occasionally necessary in such cases as undue prominence of the maxillary alveolar process, particularly when associated with a short upper lip, or when marked irregularities of the bony contour of the alveolar ridge exist ; it may also be done after multiple extractions in order to attain rapidly a smooth and even contour of the alveolar ridge for the greater ease and comfort of fitting and wearing dentures shortly after the extractions have been completed. The operation is, however, not often required.

In cases where extractions have not recently been done the operation may be done by making an incision down to the bone along the border of the alveolar ridge and raising a flap on each side with a periosteal elevator ; the excess alveolus is then exposed and may be removed with chisels and bone-cutting forceps. Some operators smooth over the bony edge after this with carborundum stones on a dental engine; any redundant gum may then be trimmed away with curved scissors and the cut edges approximated and sutured with interrupted horsehair sutures over the trimmed alveolus. Hæmorrhage is apt to be troublesome, and as some absorption may always be expected in alveolar bone it is better to remove too little than too much. In cases done after multiple extractions the division of soft tissues and laying bare of the bone is already done, and it only remains to trim off sharp alveolar prominences with small bone-cutting forceps and round off sharp edges with a carborundum stone before trimming the edges of the soft tissues so as to get good approximation before suturing them, unless of course it is desired to remove further bone on account of undue bony prominence of the alveolus.

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[1] For further details of this operation the student should consult Bowdler Henry's article in the *American Journal of Orthodontics and Oral Surgery*, 1938, xxiv, 72.

(D) THE EXTRACTION OF TEETH UNDER GENERAL ANÆSTHETICS.[1]

Nitrous Oxide, administered with air or oxygen, still holds the primary place for dental extractions for the following reasons:—

(1) The patient requires no preparation beyond starvation for three hours.

(2) The induction is speedy and not too unpleasant.

(3) Recovery is equally quick and complete, thus allowing the patient to leave the dental surgery within a short time of the extractions.

(4) Anæsthesia can be prolonged indefinitely in the average patient.

(5) Provided asphyxia is avoided nitrous oxide anæsthesia is moderately safe.

The present tendency is always to administer it by the nasal route; only in the very difficult and alcoholic patients is it necessary to resort to a mouthpiece as well as a nasal attachment, and then the former can be abandoned once the patient is induced. A pharyngeal pack composed of gauze, gamgee, marine sponge, or rubber, with a tape attachment is essential in all cases as it serves three very useful purposes:—

(1) Limits, if not entirely prevents, mouth-breathing and

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[1] For the revision of the details of anæsthetics the authors are indebted to J. H. T. Challis.

therefore assists in the maintenance of anæsthesia through the nose.

(2) Aids in preventing foreign bodies such as teeth, fillings, or splinters thereof, entering the œsophagus or respiratory tract.

(3) Can be used as a swab to dry up fossæ, or the mouth generally, of accumulating blood.

This pack is best inserted by the anæsthetist in such a way as not to impede nasal respiration or cause coughing or retching, but care must be taken that it does not obscure a clear view of the teeth to be extracted. Having inserted this pack, it is obviously the duty and responsibility of the anæsthetist to see that it is removed at the completion of the operation.

The present tendency of administering nitrous oxide and oxygen rather than nitrous oxide alone has the following advantages :—

(1) The danger of asphyxia is lessened.

(2) Anæsthesia is quieter.

(3) Congestion of the mucous membrane is lessened, and, therefore, the operator gains a clearer view of the teeth to be extracted.

The disadvantage is that patients display a tendency towards nausea and giddiness.

Ethyl Chloride is a useful anæsthetic for short dental extractions lasting up to one minute in very small children to whom the administration of nitrous oxide may be found most difficult.

It is safest given on an open mask rather than in a closed bag, although the former method is much more extravagant.

Divinyl Ethers (Vinasthane) can be used in the same way as ethyl chloride. Return to complete consciousness is perhaps slightly quicker and duration of anæsthesia shorter.

Ether is no longer used as the sole anæsthetic owing to the unpleasantness of its induction, but it is a useful adjunct to prolong and deepen a nitrous oxide and oxygen anæsthesia. Should prolonged and difficult oral manipulation be anticipated the patient must be fully prepared as for any major surgical procedure. The administration of omnopon gr. $\frac{1}{3}$, scopolamine gr. $\frac{1}{150}$ one hour prior to the induction of anæsthesia fulfils two useful purposes : allays those fears always associated with the anticipation of surgical interference, and secondly, dries up oral secretions.

The induction is performed by nitrous oxide and oxygen and sufficient ether is added to allow the easy passage of a tube into the trachea by the nasal route, thus leaving the mouth entirely at the disposal of the dental surgeon. The pharynx is well packed with gauze wrung out in saline or liquid paraffin to prevent any post-operative soreness of the throat, which is apt to occur if dry gauze be used. The administration of the anæsthetic is continued through the nasal tube. This somewhat complicated procedure is only permissible in the hands of those trained in the passing of intratracheal tubes, and, as with all anæsthetics, requires the full and undivided attention of the administrator.

Chloroform.—This anæsthetic certainly holds no place in anæsthesia for dental work unless specially ordered by a competent medical authority, and even then should never be administered except in the horizontal position.

Evipan and Pentothal Sodium.—These two drugs are administered by the intravenous route, and produce surgical anæsthesia lasting from five to seven minutes, but should it be necessary, anæsthesia can be prolonged up to half to three-quarters of an hour by giving continuous intravenous injections. Although it is a common practice to administer these drugs in the sitting position, the consensus of opinion is that under no conditions should they be given other than in the horizontal posture as they cause a heavy fall of blood pressure. Return to consciousness, although apparently quick, is not always complete for perhaps some eight to ten hours, therefore the administration of these drugs is undesirable unless the patient can be under full nursing care for some time following the injection.

The Preparation and Care during Extractions.—The last meal should be taken at least three hours before the administration of an anæsthetic and the meal should be of a light character. The position of the patient during the induction of anæsthesia and whilst under its influence should differ but little, if at all, from those already advocated for teeth extraction, except that the head should not be placed so far back. All tight clothing must be loosened, dentures removed from the mouth, and care taken to avoid any word or action likely to alarm the patient before and during the induction of anæsthesia. The operator should always decide beforehand precisely what he intends to undertake, and it is wise not to attempt too much. Care should be taken to avoid pricking the gum during the examination of any roots which are to be extracted. The prop should be placed on sound firm teeth in such a position that the operator can work without hindrance, and when the preparations are complete a final view of the mouth should be taken. The prop should be adjusted by the operator, as he will know to what extent the mouth must be opened, but at the same time the anæsthetist should see that the position of the prop is not likely to obstruct respiration. *Where several teeth have to be extracted at one sitting*, their order of removal should be decided upon before the operation is commenced, and, if any particular tooth is causing pain, it should be extracted first. As far as possible the order of removal should be arranged so that changes of instruments are reduced to a minimum. As a rule mandibular teeth should be extracted before maxillary teeth, because if the latter are removed first the blood may pass down and so obscure the mandibular ones. For the same reason roots should be removed before the whole teeth. *Each tooth or root must be cleared from the mouth before any attempt is made to remove another*, unless the gum is thoroughly adherent, when the tooth or root may be left in the mouth and freed from the gum when the patient has recovered.

During the operation of extraction the anæsthetist should :—

(1) Hold the head firmly with his hand on the forehead and his shoulder against the patient's head in order to prevent over-extension of the head, in which position there is danger of foreign bodies passing into the air passages ;

(2) Protect the lower lip from being pinched between the handles of the forceps and the mandibular teeth—a contingency which is very likely to occur when the head is over-extended ; and

(3) Clear away any blood which interferes with the operator's view.

When several teeth are to be removed on both sides of the mouth, it will be found useful to remove the maxillary and mandibular teeth on one side first, and then to cover the wounds with pledgets of wool, which should be held in place with the 'Mason' (or 'Ferguson') gag. By this means the hæmorrhage is held in check during the operation on the opposite side of the mouth.

It has been observed that in extensive dental operations students are inclined to operate with blood flowing freely over the roots that require extraction. Such a practice invariably leads to undue laceration of the gums, and considerably retards the healing of the wounds.

It is very important that after the conclusion of any operation involving the extraction of teeth *all teeth should be carefully counted and examined*, lest any tooth or portion of tooth or filling (calculus should have been removed beforehand) should have become fractured during the operation; such unaccounted-for teeth or fragments have occasionally been known to find their way into the respiratory passages or lungs in spite of pharyngeal packing, and later on give rise to serious or even fatal sequelæ. If they cannot be accounted for, either in or out of the mouth or fauces, the patient must be informed and a radiogram taken, even in the absence of any symptoms that such foreign body has found its way into the respiratory system.

Whenever a general anœsthetic is given for the removal of teeth two persons should always be present, one to concentrate his attention on the administration of the anœsthetic, the other, on the removal of the teeth. This rule should be strictly adhered to. It is impossible for one person to operate and at the same time observe the condition of the patient during the anæsthetic period.

The methods of employing local anæsthetics for the extraction of teeth are described in Chapter XXXVI.

(E) DIFFICULTIES, COMPLICATIONS AND SEQUELÆ OF EXTRACTION OF THE TEETH.

(1) Difficulties, Complications and Sequelæ connected with the Teeth themselves.

(*a*) *Undue Resistance of the Teeth and Alveolar Process.*—Certain teeth offer more than ordinary resistance to removal. The teeth of persons of strong physique are nearly always very firmly fixed. Isolated teeth also are always more firmly fixed than those in series, a circumstance which is due to the consolidation of the surrounding bone. With experience and observation the teeth which are likely to be especially resistant can to some extent be recognised. When undue resistance is encountered, steady and repeated attempts should be made to free the tooth by moving it slightly in different directions; too much force used in one direction would be certain to fracture the tooth or the alveolar process. It may even be found impossible to remove a tooth, in which case it is advisable to dismiss the patient and make an attempt two or three days later, when the tooth will probably be loose as a result of the inflammatory process set up by the previous attempts at extraction, and it can then usually be easily removed.

The causes of undue resistance are :—

(i) Abnormal density of the alveolar process.

(ii) Divergent and twisted roots.

(iii) Alteration in the shapes of the roots brought about by parodontal inflammation.

(*b*) *Fracture of the Tooth.*—The principal causes of this accident are :—

(i) The use of forceps which do not accurately fit the tooth.

(ii) The use of unnecessary or wrongly applied force in an attempt to loosen the tooth in its socket.

If a tooth has been fractured, the patient should be made to rinse the mouth until the bleeding has ceased ; the socket should be dried with cotton-wool, and the position and edge of the root defined with a probe before an attempt is made to remove the fractured portion. Neglect of these steps often leads to failure to remove the remaining portion of a fractured root. As a general rule more than two attempts to remove a fractured root should not be made at one sitting, and if the second attempt prove fruitless, the patient should be dismissed and a fresh attempt made after a period of one or two days, as the tooth will probably then be looser ; moreover, the hæmorrhage will have ceased and it will be possible to obtain a clearer view of the root. Before dismissing the patient an anodyne mouth-wash should be prescribed, and the pulp, if exposed, touched with a strong escharotic. The lower third of a root may generally be left without fear of immediate harm ; but it is always advisable to inform the patient when any portion of a tooth is allowed to remain in the jaw, as such knowledge may be of assistance should any trouble arise at a subsequent date.

When a small portion of the apex of a tooth has been fractured and remains *in situ* it is almost always lying loose from its attachments at the bottom of the socket, but it is not possible to seize it ; in such cases, if the wound be freed from blood, the pulp canal opening may be seen ; if the end of a fine Donaldson broach (multibarbed) be inserted into this opening and wriggled into it, the apex of the tooth will almost always come away on the end of the bristle when it is withdrawn.

(*c*) *Crowded and Irregular Teeth.*—The removal of these has already been referred to on p. 845 *et seq*.

(*d*) *Dislocation of a Neighbouring Tooth.*—This accident, which is generally due to a crowded arrangement of the teeth, seems to occur most frequently with the removal of the mandibular first permanent molar, the neighbouring tooth usually involved being the second premolar, which is simultaneously dislocated from its socket. To avoid the contingency the thumb should be placed on the tooth which shows a tendency to move, and in the removal of the tooth which is being extracted only as much force should be exerted as can be controlled by the thumb. If a neighbouring tooth is dislocated it must be replaced and, if necessary, secured with a ligature or splint. If the pulp subsequently shows signs of degeneration it should be removed and the canal treated.

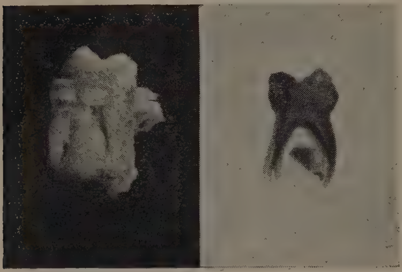
i. ii.

FIG. 892A.

i. $|E$ extracted from male aged $3\frac{6}{12}$ years having a dead and infected pulp and much periapical inflammation giving rise to adhesions between it and the tooth sac of the successional tooth.
ii. Radiogram of i. showing the partly formed crown of $\lceil 5$ *in situ*. (E. S.)

(*e*) *Removal of an Unerupted Premolar.*—This may be an avoidable or an unavoidable accident. At times the developing premolar is so firmly embraced by the roots of the deciduous molar that, during the extraction of the latter tooth, the premolar is removed; such an accident cannot be avoided. It is an avoidable accident when it occurs through using too much force in the extraction of the roots of a deciduous molar.

In quite young children the tooth sac of the premolar may be so firmly attached to the deciduous molar that its removal is unavoidable; when the deciduous molar is pulpless and infected periapical inflammation may cause adhesions between it and the premolar tooth sac (fig. 892A, i and ii).

(*f*) *Fracture of One Tooth in Extracting Another.*—In the extraction of mandibular teeth with hawk's bill forceps a maxillary tooth may be fractured. This accident, which is generally due to inexperience, is caused by the tooth which is being extracted suddenly leaving its socket in consequence of force being used in an upward rather than an outward direction. It may, however, occur when a mandibular tooth has been more than normally resistant. It is well, therefore, for the operator to be on guard by keeping the thumb or a finger of the left hand over the joint of the forceps. An adjacent tooth may also be fractured in using the elevator.

(2) Difficulties, Complications and Sequelæ connected with the Jaws.

(*a*) *Fracture.*—Accidental fracture and removal of a *small piece of the alveolar process* is not uncommon, but the result is not serious. It is sometimes unavoidable, but it may be due to placing the blades of the forceps on the outer side of the bone instead of between the bone and the root of the tooth.

Extensive fracture is sometimes seen; for instance, in a case that came under notice at the Royal Dental Hospital, an unqualified person in removing the mandibular right permanent first molar fractured the bone in a horizontal direction, so that the second and first premolars with the canine were completely separated from the body of the bone. Fracture of the maxillary tuberosity may occur during the removal of the third molar, and Main Nicol[1] records such an accident during the removal of the permanent second molar. An example of severe fracture is recorded by Cattlin,[2] where, in an attempt to remove a maxillary third molar with an elevator, the tuberosity of the maxilla, a portion of the floor of the maxillary sinus and part of the sphenoid were fractured. Fracture of the mandible due to extraction of the teeth occasionally occurs, and is most likely to happen during the removal of misplaced mandibular third molars. In a series of about two hundred cases of mandibles fractured during the removal of teeth[3] coming under the notice of one of us (E. S.) in a period of thirty-five years, all except three were caused in the removal of mandibular third molars.

Recently in removing a maxillary permanent second molar for a patient aged eighteen the thin-boned roof of the crypt of the third molar came away attached to its distal aspect, and the patient on rinsing the mouth spat out the third molar in its intact follicle; the bone of the roof of the third molar crypt is very thin and friable at this age, and probably such accident would be better known were it not for the fact that it is only comparatively rarely that permanent second molars are removed at this age.

(*b*) *Necrosis* of the alveolar process may result following extraction and is generally caused by undue violence or by the occurrence in the wound of some septic process; it occurs with far greater frequency in the mandible on account of the poor drainage. For treatment see Chapter XXX. (See also p. 925.)

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[1] *Trans. Odonto. Soc.*, xxviii, 3.

[2] *Ibid.*, iii, 138.

[3] Mostly cases that came before a professional protection society.

(*c*) *Dislocation of the Temporo-mandibular Articulation.*—The use of too much force in extracting a mandibular tooth may lead to unilateral or bilateral dislocation if the temporo-mandibular articulation has been previously dislocated or is lax. Dislocation of the jaw can easily be reduced by placing the thumbs on the molar teeth, the fingers being placed beneath the chin. A downward movement is to be used with the thumbs and an upward with the fingers. It is well to wrap some lint or a towel round the thumbs to prevent them being injured. If necessary, the leverage can be increased by placing corks or some similar wedge between the molars.

(*d*) *Opening the Maxillary Sinus.*—This accident may occur during the removal of the maxillary molars, or, occasionally, the second premolar, and may be due to a portion of the bony floor of the cavity adhering to the teeth, or to the fact that the root of the tooth has only been separated from the sinus by its lining membrane, which has been torn away during extraction. The condition is recognised by epistaxis and by fluid passing from the mouth into the nasal fossa via the sinus. The condition usually does not call for treatment unless the opening persists, a circumstance which is extremely rare, and it is probable that it is produced in minor degrees much more often than we are apt to think.

(*e*) *Forcing a Root into the Maxillary Sinus.*—This occurs mostly in connection with the extraction of the maxillary second premolar root (fig. 893) and the buccal roots of the permanent first molar. If a root has been dislocated into the cavity but partly remains in its socket, it should be left alone unless there is reason to regard it as a source of focal infection, as any attempt to remove it might only result in complete dislocation of the root into the sinus. The socket should be kept quite clean by the continual use of antiseptic washes. When a root has been forced completely into the maxillary sinus, if the opening is of some size, attempts may be made to syringe it out, using an aural syringe of 5 or 6 oz. capacity, or a Higginson syringe; the root may be evacuated in the return flow from the sinus. If this treatment fails, or the opening is a small one, it is best to allow the opening into the mouth to close up, and remove the root by opening into the antrum via the inferior meatus of the nose or by a flap operation from the buccal aspect. If an opening from the mouth into the antrum is kept open the surfaces of the opening are apt to become epithelialised over by proliferation from the epithelium lining the mouth joining with that lining the antrum ; when this has occurred, though the opening will ultimately be reduced in size by scar contraction, even till its surfaces lie in contact, there will always remain a potential opening along which infection can and occasionally does travel.

(*f*) *Forcing a Tooth into an Abscess Cavity.*—This accident should be treated by enlarging the opening into the cavity and syringing it out in the return flow, using a syringe as indicated under (*e*).

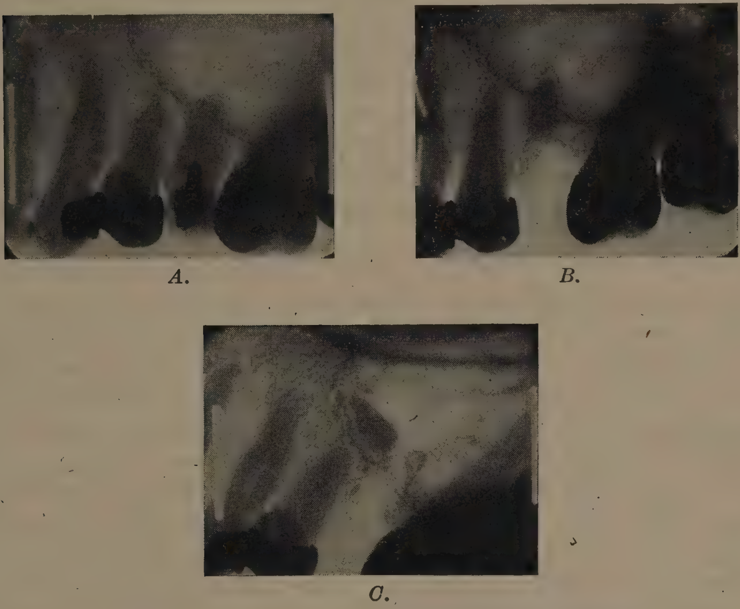
A. B. C.

Fig. 893.—Showing a maxillary second premolar root before, during and after displacement into the antrum. A week elapsed between the taking of A and B pictures, and 18 months between B and C.

(*g*) *Inability to open the Mouth.*—This renders extraction of the teeth more difficult than usual. When the closure is the result of inflammatory trouble in connection with the mandibular molars, an anæsthetic should be given and the mouth opened forcibly with a Mason's gag. If the closure is the result of tonic contraction of the muscles (trismus), ether should be used in order to overcome the resistance of the muscles, as nitrous oxide would not have the desired effect.

(3) Difficulties, Complications and Sequelæ connected with the Soft Tissues.

(*a*) *Extensive Laceration of the Gum.*—The soft tissues naturally suffer when a tooth has been difficult to remove, and may be severely lacerated when the gum attachment to the tooth is unusually tenacious. Extensive laceration occurs most frequently in connection with the removal of the mandibular third molar, but it may occur in connection with the removal of loose teeth. When the gum is more than usually adherent, the tooth should be left in the socket until the gum attachment has been divided with a pair of scissors or a scalpel. Continued attempts to remove the tooth with the forceps before the gum has been detached will only increase the laceration. Where the gums have been badly lacerated, an anodyne mouth-wash should be prescribed.

(*b*) *Wounding the Tongue.*—This is most likely to occur under nitrous oxide, as the tongue during anæsthesia is generally swollen, and is, moreover, not under the control of the patient. Wounding of the tongue is nearly always due to carelessness in the use of the elevator. When the tongue is much lacerated, the overhanging portions should be trimmed off with scissors and the surface kept clean with antiseptic mouth-washes.

If the tongue is punctured and the wound does not involve a large branch of the lingual artery, but yet bleeds freely, the tongue should be drawn forward; if this does not stop the bleeding the insertion of a stitch will generally cause the hæmorrhage to cease.

If the tongue is punctured and a large branch of the lingual artery is involved, the finger should be placed on the back of the tongue and the organ drawn forward, thus compressing the lingual artery against the hyoid bone. The bleeding point must then be sought for, and, if found, an attempt should be made to twist the wounded vessel. If this fails, cauterisation may be tried, and, as a last resource, if cauterisation does not stop the bleeding, the lingual artery must be tied.

(*c*) *Bruising the Lower Lips.*—This may occur in the removal of maxillary premolars and molars, and is due to the mouth being insufficiently opened, and to the use of forceps of too straight a pattern.

(*d*) *Injury of the Mandibular Nerve.*—The mandibular nerve at times runs in close proximity to the roots of the second molar, and more commonly to the third molar, and may be injured during the extraction of that tooth. Occasionally the nerve pierces the roots of the third molar, and extraction of the tooth then tears the nerve asunder. Montague F. Hopson possesses a specimen in which the nerve pierced the root of the permanent second molar. Loss of sensation over the parts supplied by the nerve, with dribbling saliva, generally follows the accident, but as sensation is usually ultimately restored it is evident that the nerve generally re-unites. On the other hand, if it is only bruised, there may be no loss of sensation though the pain may be intense.

(*e*) *Suppuration of the Tooth Socket.*—Suppuration in the wound caused by extraction is often due to neglect of proper precautions. It is more likely to occur when the vitality of the tissues in the vicinity of the tooth has been lowered ; for example, in cases of chronic periodontitis, or where there is a lowered general vitality, as seen in such conditions as diabetes, chronic nephritis, and tuberculosis.

Suppuration of the wound may be due to direct infection either from the mouth or from the inhalation of air laden with infectious organisms. Suppuration from the latter cause is frequently met with in those constantly working in hospital.

Where there is suppuration, the socket should be thoroughly syringed with some antiseptic, such as warm carbolic acid, 1 in 80, and kept clean by frequent irrigation. Suppuration occurs more frequently in the mandible than in the maxilla, the drainage of the maxilla being more easily effected owing to the dependent position of the sockets. When suppuration occurs in patients of diminished vitality a tonic form of treatment should be prescribed. Aperients should also be prescribed.

(*f*) *Persistent hæmorrhage following tooth extraction* is a serious complication, and one which needs prompt treatment. Persistent hæmorrhage may be due to :—

(1) Pathological changes in the artery which supplies the tooth or its socket, these changes being frequently induced by inflammation around the apex of the root, the vessel becoming adherent to its bony surroundings and thus being prevented from contracting.

(2) A defect in the process of fibrin formation causing a defect in the power of clotting.

This is found in the general condition known as *hæmophilia*. This condition is inherited by the male through the female parent; it has been shown not to occur in negro races, though it appears in half-breeds,[1] and is due to a deficiency in thrombokinase, the elements from which this substance is derived, the blood platelets and leucocytes being respectively absent and fewer in number. There is no deficiency of calcium in this disease, the quantity present (in the plasma) being relatively as great as that in normal blood.

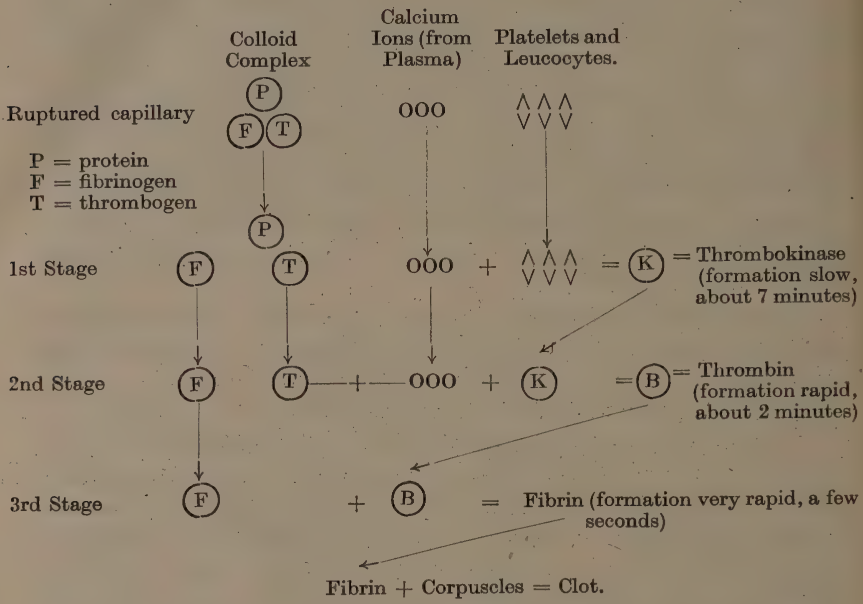
Colloid Complex
Calcium Ions (from Plasma)
Platelets and Leucocytes.
Ruptured capillary
P = protein
F = fibrinogen
T = thrombogen
P
F
T
000
1st Stage
000
K
= Thrombokinase (formation slow, about 7 minutes)
2nd Stage
000
K
B
= Thrombin (formation rapid, about 2 minutes)
3rd Stage
F
B
= Fibrin (formation very rapid, a few seconds)
Fibrin + Corpuscles = Clot.

Fig. 894.

The essential factors for clotting in freshly shed blood are:

Protein
Fibrinogen
Thrombogen (or prothrombin)
} which exist as a colloid complex.

In addition are needed:

Calcium ions (derived from the plasma).
Platelets and leucocytes.

These interact as shown in fig. 894.

In the blood of hæmophilics there is, therefore, deficient fibrin formation. The addition of leucocytes (a precursor of thrombokinase) to hæmophilic blood causes rapid clotting because the derived thrombokinase thus added completes, together with thrombogen and calcium, the factors necessary for fibrin formation. At the same time liability to hæmorrhage may exist in females even to the extent of potential female hæmophilics. Indeed, concerning hæmophilia, Armstrong Davison [1] writes :—

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[1] *Journ. Amer. Med. Assoc.*, 1935, 525.

> 'Actually the disease is inherited from male and female alike according to strict Mendelian laws, modified only by the fact that the gene in question is borne upon the X-chromosome. The gene is recessive, and thus, if a female carrier marry a normal male, the chances are (if the number of offspring be sufficient) that half the male children will be bleeders and half the female children carriers. Should a hæmophilic male marry a normal female, all the male offspring will be normal and all the females carriers. If, on the other hand, a hæmophilic male marry a carrier female the first filial generation will be composed of normal and hæmophilic males in equal proportions, and carrier and hæmophilic females, also in equal proportions. It will thus be seen that a hæmophilic female is not an extraordinary phenomenon, but is the natural, though necessarily rare, outcome of the laws of heredity.'

Other possible sources of excessive hæmorrhage which are hereditary also exist. One of these is described by Hess as an hereditary form of purpura hæmorrhagica, and by Glanzmann as hereditary hæmorrhagic thrombasthenia (hæmogenia); it occurs in both sexes, but more often in females. It has been described as a sex-linked dominant, but is almost certainly not confined to this form of inheritance. Another condition which has been confused with hæmophilia and may occur in females is that of multiple hereditary telangiectases with recurrent hæmorrhages; it has been recorded in some fifty-six families and is very regularly inherited as a Mendelian dominant.

Liability to hæmorrhage is also characteristic of certain other general conditions such as lymphatic and myeloid leukæmia, polycythæmia, pernicious anæmia, scurvy, gall stones, and purpura (a symptom, not a disease).

Many people who are not true hæmophilics show a tendency to hæmorrhage.

Bleeding is more likely to follow the extraction of teeth for parodontal disease than for other conditions, and it is possible that this is due to a chronic arteritis of the apical vessels.

Secondary hæmorrhage is due to sepsis of the tooth socket.

Treatment.—If the hæmorrhage following tooth extraction is at all profuse, it is a useful plan to give the patient some tincture of hamamelis in the water used for rinsing the mouth. Should the bleeding continue, an examination must be made to ascertain (1) whether the bleeding emanates from the gum or from the socket of the tooth; (2) whether the blood shows a tendency to coagulate. If the blood coagulates readily it is probable that the hæmorrhage is due to some condition which prevents the natural contraction of the vessels. If the blood does not coagulate readily the hæmorrhage is probably due to some defect in the process of fibrin formation.

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[1] *Brit. Med. Journ.*, 1934, 2, 1228; and also 1935, 2, 587.

If the hæmorrhage is from the gum, search should be made for any small vessels that may be the cause of it, and, if found, they should be twisted or compressed. If the vessel is only partially divided, it should be completely severed, as this will probably allow contraction and retraction to take place. If the bleeding is capillary in character, a pad of lint applied with firm pressure is usually sufficient to stop it, and a method which is efficacious is as follows: With a curved needle in a holder, sutures of horsehair are passed through the gum from one side to the other (one usually being sufficient); the ends are then tied tightly, the effect being to exert pressure on the gum and at the same time retain the clot in position.

When the hæmorrhage proceeds from the socket the following mode of procedure is adopted: The socket is first freed from clot, then syringed, then dried out with a pledget of cotton-wool; a thin strip of antiseptic gauze is next firmly packed into the socket, care being taken that the gauze reaches the end of the socket; a plug of lint is then placed over all and kept in position by antagonism with the opposing teeth, a four-tailed bandage being used for this purpose.

Certain drugs are occasionally used in conjunction with gauze packing, of which tannic acid and adrenalin are the chief; this latter drug acts by constricting the unstriped muscle fibres of the blood-vessel walls, but it must be remembered that on absorption it raises blood pressure, and so, if used freely, it may cause a tendency to recurrence of hæmorrhage later on.

If the approximal teeth are standing, an excellent method of keeping the plug in the socket is to wedge a piece of wood between them. But if the hæmorrhage is severe it is better to use the four-tailed bandage to make more certain of retaining the plug in position.

Calcium lactate should be employed, a dose of 60 grains being given, followed by three doses of 15 grains at intervals of four hours. It is best used freshly prepared, and for this purpose the following prescription is a useful one :—

℞ Acidi lactici	gr. 200
Calcii carbonas præcip.	gr. 75
Aq. chloroformi ad	℥viii

℥i = gr. 30.

In severe cases firm plugging of the socket is very important; and if the patient is unable to stand the pain a general anæsthetic should be administered.

The general directions to be given to the patient, though apparently trivial, are most important and should never be omitted. The patient should be advised to avoid all forms of excitement ; to assume the sitting position usual during the day, and to use a high pillow at night. The patient should be fed through a bent tube and all fluids should be given cold. Alcohol must be avoided.

An important factor with reference to hæmophilics, according to Paul Fildes, is that after such a patient has had a hæmorrhage, subcutaneous or otherwise, there is a phase lasting a few weeks in which the blood has an increased tendency to clot, and if an extraction is a necessity, it is best done during that period.

When the blood coagulates readily, pressure is usually sufficient to arrest the bleeding, but if the blood does not coagulate it is advisable to assist the formation of fibrin by the local application of a preparation of fibrin ferment, an efficient form being ‘ Protagulin.’ [1] To employ this drug the ‘ Protagulin ’ should be dissolved in normal saline solution, and applied to the base of the socket on a pledget of cotton-wool before inserting the plug of gauze.

J. D. Cambrook [2] has had success with and recommends the intramuscular injection into the buttock of 15 c.c. whole blood in known cases of hæmophilia before the operation is undertaken. Serum injected into a hæmophilic is said not to be very effective in controlling hæmorrhage, but fresh serum from man, horse or rabbit given intravenously is said to bring the clotting mechanism to normal within 48 hours and maintain it so for a week.

Hæmoplastin is also advocated. It is a serum derivative from equine and bovine sources and consists chiefly of thrombogen (or prothrombin), antithrombin and other thrombokinases in physiologically balanced solution.[1] It is supplied in bulbs containing 2 c.c., and in severe cases of hæmorrhage, in addition to the local application of the drug, 1 to 2 c.c. should be injected intravenously or subcutaneously, and the dose repeated every four to six hours until the hæmorrhage is controlled.

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[1] Protagulin can be obtained from Messrs. Bell and Croyden, 50 Wigmore Street, W.

[2] *Dental Record*, October, 1933.

Oil of turpentine is strongly recommended by F. St. J. Steadman.[2]

In fifty cases which required packing of the sockets the bleeding ceased within two minutes. Oil of turpentine has the advantage of being a powerful antiseptic, so that when the gauze packing is removed it still has a faint smell of turpentine, and the wounds are much cleaner than in cases where other hæmostatics are used.

The patient should be seen within twenty-four hours after treatment. If the bleeding has ceased, the plug may be removed and the wounds syringed regularly. With severe hæmorrhage the plug should be allowed to remain for two days, and if the hæmorrhage has not then ceased, the socket should be replugged. In cases of intractable hæmorrhage, hæmophilics and other conditions where there is a lessened tendency of blood to clot, the hæmostatic property of the venom of some snakes is now often used; the chief of these are the venoms of Russell's viper (*Vipera russelli*) and the Colubrine Australian tiger snake (*Notechis scutatus*); the clotting power of the latter is said to be the highest attainable and to have advantages over the viperine venom in that the latter may produce a severe gastritis and is said to render the tissues more susceptible to bacterial invasion and be proteolytic even in weak solutions, so that firm clots may soften and break down. The viperine venom is used in concentrations of 1 in 10,000 and the Colubrine venom 1 in 5000. Burgess Barnett[3] recommends the viperine venom for dental use in a strength of 1 in 10,000 on 'a piece of wool shaped like a mushroom with a very short, thin stalk, used so that the pressure on it squeezed the venom solution into the cavity, and its removal subsequently did not disturb the clot.' With clean sockets bleeding seems to be controlled almost immediately, but if sepsis is present replugging may be necessary. In uncontrollable hæmorrhage from the maxilla, digital pressure on the common carotid opposite the transverse process of the sixth cervical vertebra may be tried; should this fail to stop the hæmorrhage, ligature of that vessel must be resorted to. In one case of hæmorrhage from the region of the mandibular right third molar, the lip was divided in the median line and the cheek reflected from the jaw. The mandibular canal was then laid open by excising the outer plate of the bone, and the bleeding was arrested by plugging the mesial and distal ends of the canal.

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[1] Hæmoplastin is prepared by Messrs. Parke, Davis and Co.

[2] *Brit. Dent. Journ.*, xliii, 299.

[3] *Proc. Roy. Soc. Med.*, xxviii, 1469, and *Lancet*, 1934, 2, 985. See also *Amer. Journ. Med. Sci.*, 1935, 190, 779.

In extreme cases with signs of collapse a blood transfusion should be given. Cases of death from hæmorrhage following tooth extraction have been reported.

With patients predisposed to hæmorrhage extraction should be avoided if possible; but if the removal of the tooth be absolutely necessary, prophylactic treatment should be pursued for two days before the operation, by the administration of calcium lactate in doses of 15 gr. three times a day. The tooth should be extracted in the early morning, so as to have the whole day for treatment, should hæmorrhage occur. A dose of 60 gr. of calcium lactate should be administered at the time of the operation and the socket plugged at once as a preventive measure, for, be it remembered, it is far easier to prevent the hæmorrhage from occurring than to arrest it when once it has commenced. Should the bleeding prove to be uncontrollable, transfusion of blood from a donor of the same blood group or a universal donor must be carried out in order that the substance necessary to produce thrombin may be supplied.

(*g*) *Injury of the Arteries in the Neighbourhood of the Teeth.*—Wound of the lingual artery has been referred to under the heading of 'Injuries to the Tongue.' Laceration of the ranine, anterior and posterior palatine arteries may also occur. Such accidents are usually the result of the forceps slipping and are therefore avoidable.

Treatment consists in applying pressure, or in twisting or tying the divided vessel. In the case of the anterior or posterior palatine artery it may be found necessary to plug the foramina which give passage to these vessels.

(*h*) *Pain following Tooth Extraction.*—The causes giving rise to pain following the extraction of a tooth are:—

(1) Incomplete extraction of the tooth, more especially when the remaining portion contains an exposed pulp.

(2) Too rapid closure of the orifice of the socket. It sometimes happens that the opening of the wound left after extraction becomes occluded very early by clot; when this occurs the discharges from deep infection are retained and increase the inflammatory reaction in the surrounding tissues.

(3) Suppuration in the tooth socket.

(4) Extensive laceration of the hard and soft tissues in the neighbourhood of the socket.

(5) Septic osteitis is a common cause of pain following extraction and of persistent tenderness of the bone. In teeth which have been the seat of chronic periodontitis or parodontitis, the surrounding bone is frequently infected, and although the infection is held in check by the local tissue reactions, the barrier is broken down by the operation of extraction and the infection spreads. The severe earache and neuralgia which may follow extraction of the mandibular molars is due to infection of the nerve sheath and is a neuritis.

(6) Necrosis of the socket of the tooth.

(7) The presence in the wound of a foreign body. A curious example came under notice a few years ago. A patient applied for the extraction of the left permanent first molar. During the operation a portion of one of the cusps disappeared; a search proved fruitless, and it was supposed that it had been removed in rinsing the mouth. For the next three weeks the patient complained of slight pain in the socket, for which remedies were tried without success. Eventually the patient discovered the cusp on the top of the granulation tissue which had filled up the socket. In another case of the same character which came under notice the offending material was a piece of an amalgam filling. A broken portion of a blade of forceps may be the offending body.

(8) Injury to the nerve. Direct injury to the trunk of the nerve is more likely to occur during extraction of the mandibular third molar than with any other tooth. It is probable that some obscure cases of pain following tooth extraction are due to exposure and irritation of the nerve at the apex of the socket. An interesting case of this character was reported by Storer Bennett. The patient, a lady aged twenty-three, had had the maxillary third molar dislocated through the use of a Mason's gag, and as it was considered hopeless to restore the dislocated tooth it was extracted without difficulty. The socket, in spite of treat-

ment, remained painful for the next twelve days, but in the meanwhile granulated healthily, except at its apex, where by the aid of a mirror and probe a spot about the size of a pin's head was noticed, which caused the greatest agony on being touched. Incision of the nerve produced permanent relief.

When pain follows tooth extraction, treatment must obviously depend in a great measure on the cause. A thorough examination of the socket should be made with probe and mirror. When due to incomplete extraction another attempt, if considered advisable, may be made to remove the tooth, and, if this is unsuccessful, the socket should be swabbed with an anodyne drug, and, if there is an exposed pulp in the remaining portion of the tooth, the pulp should be touched with strong carbolic acid. The patient should also be advised to use fomentations.

Where the orifice of the socket is occluded too rapidly, the clot must be removed, the socket syringed out, and a small tent of lint allowed to remain in the orifice for about twelve hours. An antiseptic mouth-wash should also be prescribed.

In pain following extensive laceration and bruising of the soft tissues a mouth-wash containing chloride and sulphate of zinc is useful.[1] The pain of osteitis is difficult to allay; regular irrigation of the tooth sockets with warm 1 in 100 carbolic acid and the use of fomentations, together with the administration of aspirin in doses of 10 gr., is the most satisfactory method to adopt.

(*i*) *Traumatic Emphysema.*—A case in which traumatic emphysema followed tooth extraction is recorded by A. Turnbull.[2] A bugler sounded his bugle immediately after having had a maxillary premolar removed, and as a result his face became considerably swollen in consequence of the passage of air through the empty tooth socket into the cellular tissues.

A somewhat similar case is recorded by H. T. Binns.[3] A male, nearly sixty, had $\underline{765\,|}$ removed under local anæsthesia; immediately afterwards he said his face felt puffy but there was no obvious

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[1] ℞

Zinci sulphatis	gr. viii.
Zinci chloridi	gr. vi.
Morphinæ acetatis	gr. ii.
Aquam	ad ℥viii.

M. Fiat lotio.

To be used with an equal quantity of water as a mouth-wash.

[2] *Brit. Med. Journ.*, May 5, 1900, 1131.

[3] *Brit. Dent. Journ.*, lviii, 21.

swelling; later that evening there was considerable swelling on the side from which the teeth had been extracted, extending 'from the superior insertion of the temporal muscle and the right lower eyelid to the clavicles and on either side of the trachea, causing difficulty in swallowing but only slight difficulty in breathing, and on palpation giving a crackling sensation.' There was no pain from the wounds or swelling. The swelling reached its maximum twenty-four hours after the extractions and at the end of five days could no longer be detected. The patient's wife thought she remembered him trying to blow through a choked pipe, but beyond that history was negative. Subsequent extractions done in a similar manner were uneventful.

Another comparable case recorded by J. E. Shaefer and P. E. Williams[1] occurred following prolonged efforts in the attempted removal of a mandibular second molar; it was thought that the emphysema here probably resulted from the use of compressed air to dry the field of operation. The area affected by the swelling was bounded above by a line from the alæ of the mouth to both ears and below reached almost to the nipples.

(4) Difficulties, Complications and Sequelæ arising during Extraction under Anæsthetics.

(*a*) *Tongue Slipping Back.*—During extraction under anæsthetics the tongue, not being under control, may slip over the larynx, or may be forcibly pushed back by the fingers of the operator. Symptoms of difficult breathing or even arrest of respiration will follow this accident. Treatment consists in pushing the mandible forward with the thumbs behind the angles or pulling the tongue forcibly forward with a suitable instrument.

(*b*) *Forcing out a Tooth with a Prop or a Mason's Gag.*—This accident may arise from resting a prop upon teeth which are loose, or from placing it in such a position that undue leverage is brought to bear upon a tooth. It is an accident most likely to occur when the prop is fixed upon the front teeth and the mouth is opened to its widest extent. Under such conditions undue leverage at right angles to the long axis of the tooth is brought to bear upon the palatal surfaces of the maxillary teeth, and they are consequently forced outwards.

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[1] *Dental Cosmos*, 1933, lxxv, 377.

The accident may arise from clumsiness in the use of a Mason's gag, and great care should therefore be exercised when using this very powerful instrument. If a tooth is forced out it should, if possible, be immediately replaced and a splint applied.

(*c*) *Passage of a Foreign Body through the Isthmus of the Fauces.*—A foreign body, such as a tooth, a broken piece of forceps, or a prop, passing through the isthmus of the fauces may become impacted in either the air or food passages.

(i) In the air passages it may lodge (*a*) over the entrance of the larynx, (*b*) in the larynx, (*c*) in the trachea or bronchus.

(ii) In the food passages it may lodge (*a*) in the pharynx, (*b*) in the œsophagus, (*c*) at the pyloric opening of the stomach.

(i) *In the Air Passages.*—Should the foreign body lodge *over the entrance of or in the larynx*, the patient will be seized with a violent fit of coughing which may expel it, but if it is not expelled symptoms of asphyxia will supervene. Treatment: the head should immediately be brought forward and the finger inserted along the side of the mouth into the pharynx, and then given a forward sweeping movement; by this means the foreign body, if lodged at the back of the tongue, will probably be removed. If the foreign body is not dislodged by this method, laryngotomy should immediately be undertaken and carried out promptly, as any delay diminishes the chance of saving the patient's life.

A foreign body in the trachea or bronchus usually gives rise to a violent fit of coughing with signs of impending asphyxia. These signs pass away and are followed at intervals by fresh attacks of coughing and eventually by symptoms of collapse of the lung or lungs.

In emergency, tracheotomy may have to be performed; for the details of this operation both above the isthmus of the thyroid and below, and the operations of laryngotomy and cricotomy, the student is referred to a text-book on General Surgery.

When there is a suspicion that a tooth or a piece of a broken instrument may have passed into a bronchus, prompt measures must be taken. In the first place an examination must be made with X-rays, and this, even if negative in character, should be followed by a thorough examination of the chest by auscultation and percussion. In the event of the foreign body being lodged in a bronchus, steps should be taken to remove it; in the absence of signs or symptoms there is no immediate hurry except when the obstructing object is a vegetable substance; non-vegetable objects may remain in the lung for years without producing symptoms, but lung abcesses or bronchiectasis will occur eventually. This removal was formerly an extremely difficult undertaking, but the introduction of the bronchoscope has greatly facilitated the removal of such foreign bodies.

(ii) *In the Food Passages.—A foreign body impacted in the pharynx* will give rise to pain, symptoms of dysphagia, and dyspnœa. The patient generally has a hacking cough. Should a foreign body be suspected in the pharynx, its presence can usually be ascertained by digital exploration; this failing, the cavity should be examined by the aid of a laryngoscope.

An attempt should first be made to remove the body with the fingers; and if this is unsuccessful pharyngeal forceps must be called into use. Where the impaction is very firm, it may be necessary to perform pharyngotomy.

A foreign body in the œsophagus will cause dysphagia, and will probably give rise to constant pain; if it is situated in the upper part it will in all probability give rise to dyspnœa. On applying the stethoscope over the region of the œsophagus, a gurgling sound will be heard when the patient swallows fluids. The presence of a foreign body may be definitely ascertained by the use of the X-rays. If impacted in the upper part of the œsophagus an attempt may be made to remove the impacted body with forceps; this failing, œsophagotomy must be performed. If lodged near the cardiac end of the œsophagus an attempt may be made with a bougie to push the foreign body into the stomach; this failing, gastrotomy should be performed.

If a foreign body becomes impacted at the pyloric opening of the stomach, it will give rise to gastric dilatation. In such circumstances the stomach must be emptied of its contents, and gastrotomy then performed.

(5) Miscellaneous Difficulties, Complications and Sequelæ.

(*a*) *Menorrhagia.*—A case is recorded in which a married woman, aged twenty-five, unipara, was seized with violent bleeding from the uterus after the removal of teeth under chloroform anæsthesia. Packing the uterus with tampons saturated with various solutions, and the administration of ergot and strychnine, were tried without success. Finally the uterus was curetted and packed, the hæmorrhage ceasing at the end of twenty-four hours. An examination of the scrapings from the uterus showed the presence of chronic follicular endometritis. Menorrhagia is liable to occur with such local conditions as endometritis, and it is possible that the extraction of the teeth, combined with the chloroform anæsthesia, may have been indirectly responsible for the menorrhagia. The lesson to be learnt from this case is the necessity of avoiding dental operations, as far as possible, during the menstrual period.

(*b*) *Abortion.*—One of us has known of several cases of abortion following on multiple extractions of teeth under anæsthesia, and though such cases are rare they should act as a reminder that it does not require much in the way of a stimulus to start uterine contractions in some women, and that unless much sepsis or pain is present it is better to temporise with some alternative and conservative treatment rather than take an unnecessary risk of this possible, though fortunately rare, sequel.

(*c*) *Shock.*—The fact that tooth extraction is a surgical operation and may be followed by shock is often overlooked. As a rule, the shock which follows tooth extraction is negligible, but at times it may be well marked, especially in the weak. The effects of shock are often not sufficiently taken into account when a question arises as to the number of teeth to be extracted at one sitting. It should clearly be borne in mind that a strong, able-bodied person can bear more severe operations than one of weaker physique.

(*d*) *Syncope at the time of the operation sometimes occurs.*—In these circumstances, the operator should immediately desist until recovery ensues. Fainting is best treated by bending the head down towards the knees, at the same time loosening anything tight about the neck and waist and applying ordinary salts of ammonia to the nose. In severe cases the patient should be removed from the chair and laid on the floor, and the chest should be exposed and flipped with a towel dipped in cold water. In more severe cases it may be necessary to inject a cardiac stimulant. Fatal syncope following tooth extraction has occurred. In one case recorded [1] the patient was a female, and an attempt was made to remove a tooth, but owing to alarming syncope the operation was abandoned. A second attempt was about to be made when fatal syncope ensued. *Post-mortem* examination showed nothing beyond a slight amount of cerebral congestion.[1]

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[1] *Brit. Dent. Journ.*, vii, 32.

(*e*) *Epilepsy.*—When a person is predisposed to epilepsy an attack often commences immediately after the extraction of a tooth. If a fit occurs, the patient should be removed from the chair and placed on the floor, the clothes at the same time being loosened and a wedge of wood or some suitable material placed between the teeth to prevent injury to the tongue.

(*f*) *Inhibition of the Salivary Glands.*—A case has been recorded by G. L. Curnock[2] of inhibition of the salivary glands following the removal of a difficult mandibular molar. Within twenty-four hours there was great pain, the socket of the tooth was extremely septic, and the flow of the saliva had apparently ceased. The cause appears to have been psychical, as the operation had been dreaded, and a somewhat similar condition had followed on the occasion of an air-raid.

(*g*) *Septic and Infective Sequelæ.*—In the description of acute local periodontitis (p. 450), attention was drawn to the spread of the infection from the teeth to the cellular tissues of the face and neck. Mention was also made of the infection of the bone which is often present in parodontal disease, an infection which must be regarded as latent. The removal of these chronically infected teeth is sometimes followed by the development of an acute general infection. The surrounding soft tissues swell rapidly, the wounds become extremely septic, and symptoms of general infection appear.

Four cases of this character are recorded by W. G. Spencer,[3] and he attributes the condition to an acute infection of the raw surfaces of the gums and bare alveoli from sepsis around the mandibular incisors. It seems, however, more likely that the latent infection in the bone is started into activity by the operation.

Similarly, when general symptoms, such as pains in distant joints, are caused by infection originating from dental foci, the removal of teeth often causes a recrudescence or lighting up of the symptoms, sometimes to a severe or serious extent; it is therefore a wise thing, when called upon to remove a large number of teeth in a patient with dental infection—and particularly when general symptoms are caused thereby—to feel one's way carefully and extract one or two teeth only at first so as to note the nature and severity of the reaction it gives rise to, if any.

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[1] See also case recorded in *Brit. Dent. Journ.*, xvii, 385.

[2] *Brit. Dent. Journ.*, February 1, 1918, 75.

[3] *Ibid.*, January 28, 1922, 132.

Thrombosis is a rare sequela, and would seem to be more likely to occur in cases where it has been necessary to arrest bleeding by plugging. An instructive case was recorded by S. P. Mummery.[1] A man, aged seventy-two, had fifteen teeth removed at one sitting under nitrous oxide and oxygen, the bleeding ceasing soon after the operation. The teeth were firm and the alveolar process thickened. The same evening there was an outbreak of hæmorrhage, which was arrested without difficulty; the following day there was a slight general oozing from the sockets, and the next day a fresh outbreak of bleeding which was arrested by plugging. On the fourth day the bleeding broke out again and fresh plugs of adrenalin gauze were inserted. The temperature and pulse remained normal and the mouth clean. On the eighth day the temperature suddenly rose to 104° F., signs of thrombosis of the vessels of the arm appeared, and death occurred within the next twenty-four hours. *Post-mortem* examination revealed a small carcinoma in the prostate, some secondary deposits, and considerable thrombosis in many of the large veins.[2]

Septicæmia following extraction of teeth is rare, but cases occasionally occur; dental treatment consists in keeping the mouth wound as clean and free from infection as one can so that the entry of further organisms into the blood stream is prevented as far as possible.

Where septic and infective conditions follow the extraction of teeth it would be unfair to attribute the infection to the actual operation as a matter of course, as in many cases the infection is unquestionably caused by the neglected condition of the extracted teeth. Infection can, however, at times be directly traced to the operation, and once again attention is drawn to the urgent necessity of adopting aseptic precautions. Cases of syphilis acquired through the use of infected forceps are recorded.

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[1] *Proc. Roy. Soc. Med.* (Odonto. Sec.), vii, 84.

[2] See also case recorded by J. W. Tomlinson, *Dental Record*, xl, 314.

CHAPTER XXVIII

Replantation, Transplantation, and Implantation of the Teeth.

Replantation is the operation of replacing in its socket a tooth which has been partly or completely dislocated.

Transplantation is the operation of transferring a tooth from its own socket to that of another tooth. The transfer may be made in the same mouth, or from one mouth to another.

Implantation is the operation of forming an artificial socket in the bone for the introduction of a natural tooth.

Of these three operations, the only one which is performed to any extent in this country is replantation, the other two being of questionable utility.

(A) REPLANTATION.

Replantation should be performed only in cases of traumatic dislocation.

The operation consists in gently rinsing the tooth in a solution of some antiseptic, and replacing it in the socket with firm pressure, the alveolar process being moulded around it with the fingers. The tooth may be kept in place by a metal splint similar to that recommended on p. 167. If the patient is not seen for some hours after the accident, the pulp must be removed, and the canal filled before the tooth is replaced.

The union after replantation is brought about through the periodontal membrane. If the tooth is living and is replaced immediately, the vitality of the pulp is sometimes re-established. Magitot, who has recorded a large number of cases of replantation, maintains that the operation cannot be successful unless there is a complete ring of healthy membrane on the tooth.

The results of replantation depend upon the nature of the condition for which the operation is undertaken. If the tooth is replaced immediately after dislocation a successful result may be anticipated; but if replacement is delayed the prognosis is naturally not so good.

If a tooth has been completely dislocated and has lain on the ground, the possibility or probability of infection with the tetanus organism must be considered, particularly as this organism is an anærobe; this factor alone will militate against an attempt at replantation being made in many cases where otherwise it might be successful. An account of a remarkable case of successful replantation has been sent to one of us by E. R. Cozens, who writes concerning a girl aged eighteen years: 'her mother said that when she was ten years old she fell down and knocked her maxillary left first incisor out. Her father, who was there at the time, picked the tooth up and placed it back in the socket. Three times during that day the tooth fell out again, once when she was having her tea, and each time it was placed back again. When she went to bed that night her mother was afraid that she might swallow it, so she tied a piece of string round the tooth and then round the girl's neck. After the first day it never fell out any more and the tooth to-day looks quite normal in colour, and no different from the surrounding teeth. I tested the tooth for thermal changes and it reacted to heat and cold. The tooth feels quite firm in the socket.'

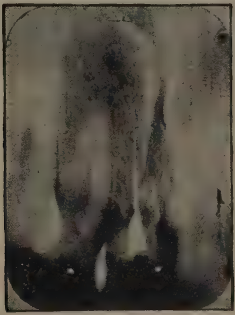

FIG. 895. (From *Brit. Dent. Journ.*)

The results, however, are not always quite so ideal. P. Pincus[1] records the case of a medical man who, at the age of seventeen, had a maxillary right first incisor completely dislocated when playing football; the tooth was replaced and held in position with wire ligatures, the pulp canal apparently being filled with gutta-percha. Ten years later the maxillary and mandibular incisors did not meet on occlusion, and the patient is certain that the incisal edges of the maxillary first incisors were symmetrical immediately after treatment. Radiological examination showed the root of the replanted tooth apparently replaced by bone (fig. 895), though the length of the root filling shows that the root had probably been of normal length at the time of the accident. The tooth appeared quite firm, but shortened (fig. 896).

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[1] *Brit. Dent. Journ.*, lx, 341.

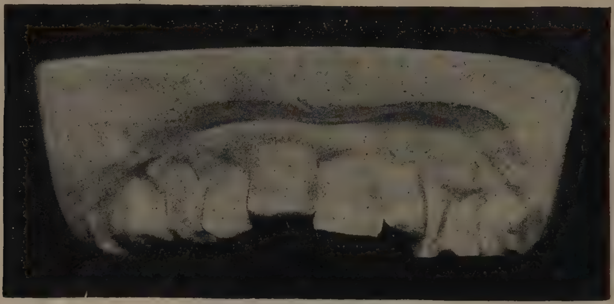

FIG. 896. (From *Brit. Dent. Journ.*)

(B) TRANSPLANTATION.

There are three distinct objections to the operation of transferring a tooth from one mouth to another: (1) The risk of failure; (2) the risk of inoculation; (3) the moral objection. Failure of the operation may be due to (*a*) want of adaptability of the tooth to its new socket; (*b*) morbid conditions of the new socket. The risk of inoculation is a strong argument against this operation—a case of transmitted syphilis has been recorded within recent times. The moral objection to the operation is also weighty, and all circumstances considered, transplantation of a tooth from one patient to another is to be condemned. The method recommended for carrying out the operation is as follows: The patient to receive the transplanted tooth is first operated upon, as little injury as possible being inflicted, and the bleeding from the socket arrested as far as possible. The tooth to be transplanted is next removed from the other patient and immediately transferred to the vacant socket and forced well into place. The transplantation of a tooth from one socket to another in the same mouth is occasionally useful, as, for example, the transfer of a healthy second incisor which has erupted internal to the arch to the socket of an unsavable first incisor.

Union in transplantation may be a process similar to that which takes place in replantation; or the process may be similar to that which occurs in implantation, viz. absorption in the first place of portions of the root and the subsequent formation, in the excavation, of bony tissue continuous with the alveolar process, the tooth thus becoming anchylosed to the jaw. In other cases, it seems possible that there is only a fibrous union.

(C) IMPLANTATION.

Implantation is an operation of more recent date than either replantation or transplantation, and is probably less justifiable even than transplantation. W. J. Younger, who introduced implantation, is very circumspect in choosing patients for this operation, and never performs the operation unless the patient is young and healthy. The tooth to be implanted must possess a healthy periodontal membrane.

The method of procedure is as follows: A crucial incision is made over the situation intended for the implanted tooth; the flaps of periosteum are then dissected up, and, by means of specially designed trephines and burs, a fresh socket is made. The socket is then syringed so as to remove all debris, and the bleeding is then arrested, the tooth being fixed into place and, if necessary, secured with ligatures or other suitable means. Some operators force the tooth into place with a hammer; in this way the tooth is firmly fixed and ligatures are not required. Younger does not always use freshly extracted teeth to perform implantation, but in some cases employs dry teeth.

The union after implantation probably takes place in a manner similar to that following many cases of transplantation. A. Amoedo advocates slight decalcification of the roots before implantation. The root is placed in a 10 per cent. solution of hydrochloric acid for from three to four hours until the surface of the cement is slightly softened, the acid being afterwards neutralised with ammonia. The results obtained from implantation are not encouraging, and do not justify the operation.

CHAPTER XXIX

Fractures of the Jaws.

THIS subject may conveniently be considered under three heads :—

(A) Fractures of the jaws.
(B) Gunshot injuries of the jaws.
(C) Pathological fractures.

(A) Fractures of the Jaws.

(1) FRACTURES OF THE MANDIBLE.

Fractures of the mandible are more common than fractures of the maxilla, and this is no doubt due to the fact that the mandible is in the more exposed position. The fractures occur more often in the body of the bone than in the ramus ; in the body of the bone they are practically always compound, in the ramus they are usually simple. The bone is generally fractured in one place only, but multiple fractures are not uncommon.

In a few cases of fracture a portion of a tooth is situated in either fragment.

(α) *Causes.*—Fractures mostly result from kicks, a blow of the fist, or a fall (especially on the chin). Undue violence in the extraction of a tooth, particularly misplaced mandibular third molars, may fracture the body of the jaw, but rarely causes a direct transverse fracture (see p. 869). Fracture of a small portion of the alveolar process during extraction is a common occurrence, but as it leads to nothing more serious than the exfoliation of the portion fractured, the injury is of little importance.

(β) *Position.*—The position of the fracture depends on the locality in which the blow is received. The most common position of fracture is in the neighbourhood of the **premolars.** A fracture caused by a blow on the front of the chin a little to one side of the mid-line is often situated in the **region of the canine** on the opposite side ; the explanation used to be that the depth of the canine socket weakens the bone in this region, but fractures commonly occur in this region in edentulous people. Another and more satisfactory explanation is as follows: The resistance to a blow on the side of the chin is met at the condylar articulation of the opposite side; the segment of bone between the regions of impact and the resisting area is a curve; the force of the blow increases the convexity of the curve and the bone gives way at the point of greatest convexity, which is usually between the canine and first premolar. If a blow is received under the middle of the chin the resistance is met at both condyles and the **neck of the condyle** is fractured on one or both sides; in a few cases the neck of the condyle is driven into the condyle, causing an impacted fracture. In the case of young children a fall on the chin may lead to a transverse fracture at the **symphysis.** Fracture in the neighbourhood of the **angle** is usually due to direct violence. Fractures of the **ramus** are generally the result of great violence and may occur in almost any position.

(γ) *Signs and Symptoms.*—With fractures in the body of the bone the symptoms are generally well marked, but with fractures of the ramus the symptoms are often obscure. In either case the patient usually complains of **pain** on opening the mouth. In the former there will be crepitus, **mobility in the continuity of the part,** and, frequently, alteration in the line of the teeth, due to **displacement.** The pain in cases of fracture is generally referred to the injured area, but in fractures about the angle the pain is at times referred to the mental foramen. In fractures of the portion of the mandible lying between the inferior dental foramen and the mental foramen, cutaneous anæsthesia is usually present over the area supplied by the mental nerve. There is often more or less salivation and, when the displacement is great, there is sometimes inability to close the mouth. In fractures of the horizontal ramus F. Coleman has pointed out that a practically constant sign is an extravasation of blood into the tissues of the floor of the mouth adjacent to the site of fracture; this becomes raised, forming a bluish, tense and elongated swelling under the side of the tongue, and is almost invariably present within an hour or two of the injury.

Radiograms should be obtained in all cases of suspected fracture.—It is advisable to obtain both antero-posterior and lateral views, as the **interpretation of radiograms** of the jaws is rendered difficult by the shadows on the plate of the skull and the vertebræ, and errors can easily be made. For example :—

(i) If the fragments slightly overlap, the line of fracture will be masked in the lateral view; the antero-posterior view will, however, show the line quite clearly.

(ii) Lateral views of the ramus are often obscured by shadows of the vertebræ and the shadow of the intervertebral discs may simulate a line of fracture.

(iii) In antero-posterior views of the incisor region the shadow of the intervertebral disc may be mistaken for a horizontal fracture.

(δ) *Displacement.*—The displacement will vary according to the character and position of the fracture, and will be slight in some cases and well marked in others.

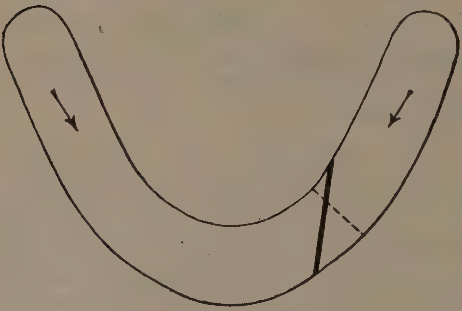

FIG. 897.

The arrows indicate the direction of force exerted by the external pterygoids.

In the **incisor region,** as a rule, no displacement occurs with vertical fractures in the region of the first incisors because the depressors and elevators of the mandible counterbalance one another. The 'slewing' action of the external pterygoids obviously cannot operate with vertical fractures. But the farther the line of fracture is removed from the median line of the mandible the greater will be the tendency to displacement. The digastrics, with the genio-hyoids, are attached to the inner aspect of the bone on either side of the median line; consequently, the depression of the larger fragments by these muscles tends to increase as the seat of fracture is removed farther from the median line.

When the fracture runs obliquely through the bone, the action of the external pterygoids will cause the fragments to overlap. Thus, in the left **canine region,** if the fracture runs through the bone from within outwards and forwards, the tendency will be for the left external pterygoid to force the smaller fragment towards the right, and for the right external pterygoid to force the greater fragment to the left, and the combined action will cause the fragments to slide one over the other, the lesser moving in front of the greater. The action of the external pterygoids is diagrammatically shown in fig. 897. The muscles which close the jaw will have a tendency to draw up the lesser fragment because there is no opposition from any of the depressors of the jaw; the depressors are all attached to the greater fragment and depress it, the right and left depressors combined exerting more force than the counteracting right elevators.

In double fractures through the canine region, with the lines of fracture taking oblique directions, the following displacement may occur: Attached to the central fragment are the digastrics, the genio-hyoids and the genio-hyoglossi, and the action of these muscles is to depress the fragment and cause it to fall over towards the tongue; the deformity thereby produced may bring about a fatal asphyxia by the falling back of the tongue if a patient in this condition is lying supine—as on a stretcher; they should therefore lie prone. The elevators of the mandible are attached to the lateral fragments and draw them slightly upwards; the left external pterygoid tends to draw the left fragment to the right and the right external pterygoid tends to draw the right fragment to the left, the result being that the lateral fragments move towards the median line and the inward displacement of the central fragment is increased (see fig. 898). In reducing fractures of this kind the foregoing facts should be kept clearly in mind.

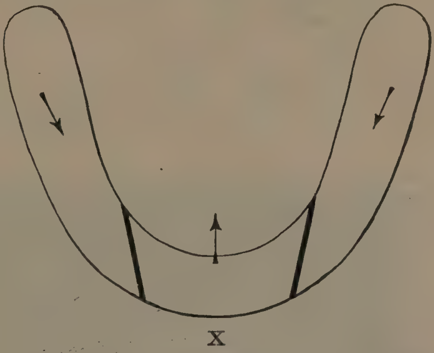

Fig. 898.—(X) Central fragment to which the depressors of the mandible are attached. The arrows indicate the direction of force exerted by the external pterygoids.

With fractures in the **molar region,** the anterior fragment is drawn downwards by the depressors of the mandible. The displacement of the posterior fragment is governed by the resistance to the upward pull of the elevators of the mandible. This resistance depends upon (i) the presence or absence of teeth and (ii) the direction of the line of fracture. If teeth are present in the posterior fragment as well as in the maxilla, the upward pull is resisted irrespective of the direction of the line of fracture. If the fragment is without teeth or possesses only teeth which are unopposed by maxillary teeth, a displacement upwards and forwards will occur, provided that the line of fracture does not resist the movement. To make this statement clear, attention is drawn to the diagram (fig. 899). If the break takes the form (*a*), (*b*), or (*c*), the upward and forward pull is resisted by the anterior fragment, but when the break is as shown in (*d*), there is no resistance to the upward and forward movement and considerable displacement follows (see fig. 900).

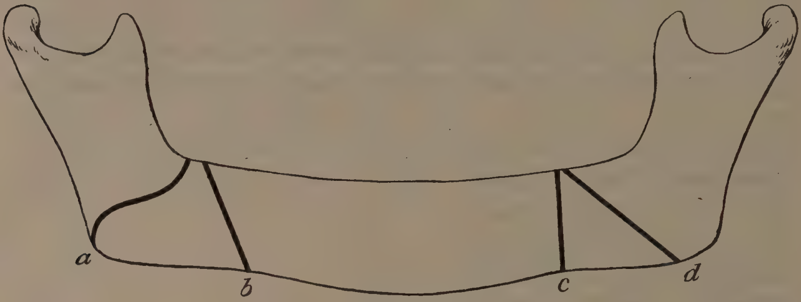

FIG. 899.

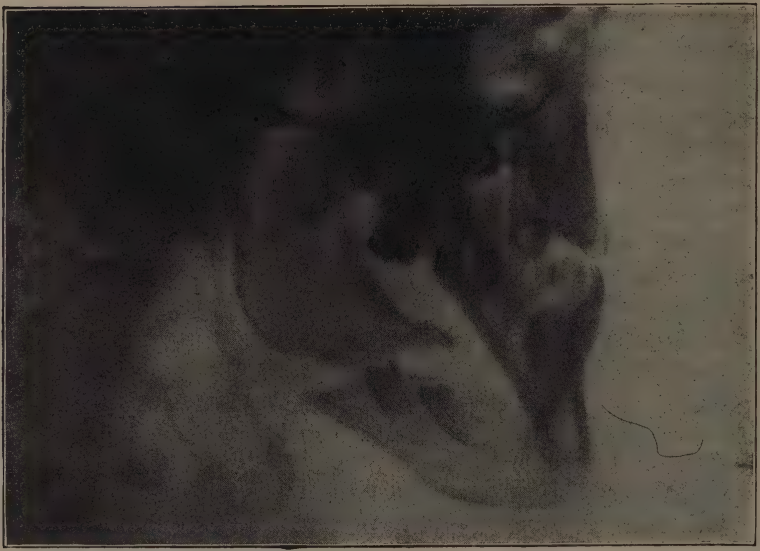

FIG. 900.—Fracture through the region of the second molar, showing marked upward displacement of the posterior fragment.

An instructive case is shown in figs. 901 to 903. The jaw was struck in the locality of the left molar teeth. The bone was fractured on the left side about the region of the third

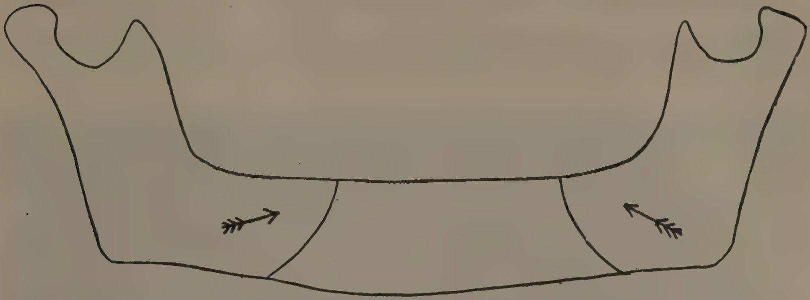

FIG. 901.—The arrows indicate the direction of force exerted by the muscles attached to the posterior fragment. (From *Brit. Dent. Journ.*)

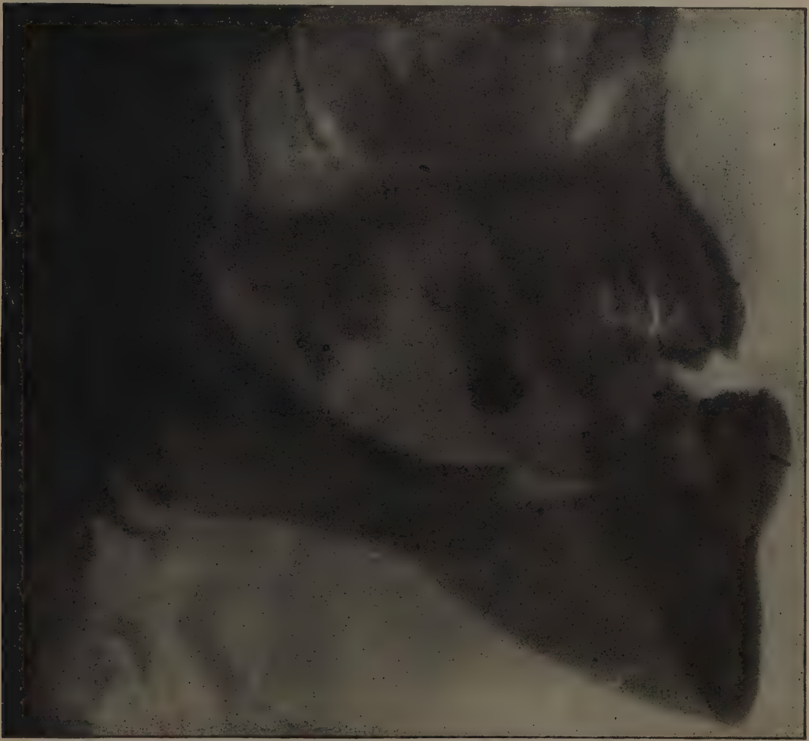

FIG. 902. (From *Brit. Dent. Journ.*)

molar and on the right side in the position of the premolars. In both fractures the break of continuity took the form of a curve, as shown in the diagram (fig. 901). Both the posterior fragments, which were without teeth, swung forwards and slightly upwards, following the direction of the curves, the combined forward movement pushing the central fragment well forwards so that the patient presented a condition of inferior protrusion. The radiograms of this case are shown in figs. 902 and 903.

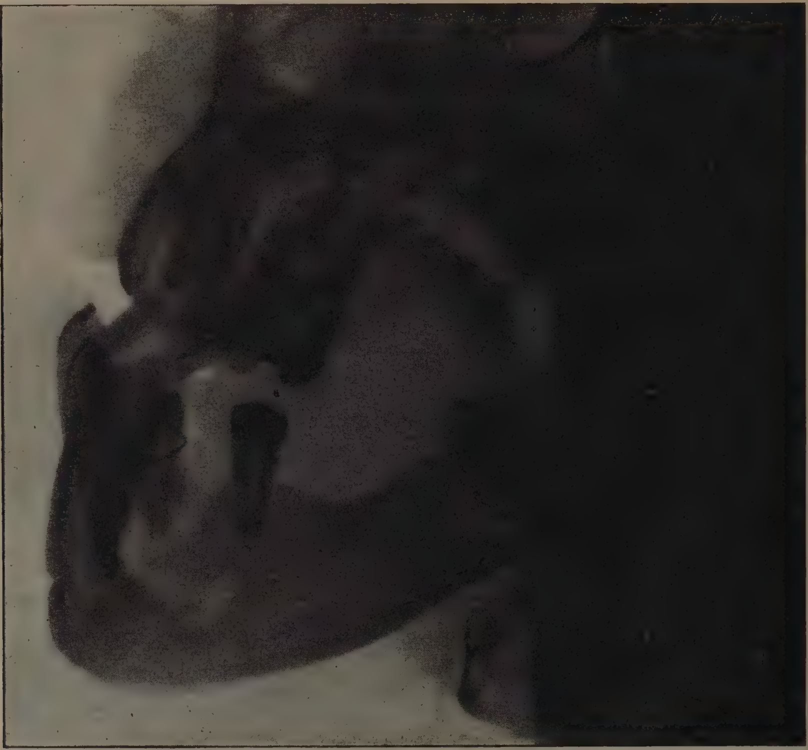

FIG. 903. (From *Brit. Dent. Journ.*)

Fractures in the molar region are often due to direct violence, the force of the blow driving one of the fragments inwards. In such cases the forward movement of the posterior fragment and the deviation of the anterior fragment to the injured side result in an overlap of the fragments which at times is extremely difficult to control in consequence of the action of the superior constrictor of the pharynx. This muscle has an attachment to the mandible immediately above the posterior extremity of the mylohyoid ridge, and each act of swallowing exerts an upward and inward drag on the posterior fragment. When the posterior fragment lies internal to the anterior, the 'drag' can be observed if the patient is made to close the mouth and swallow. When the posterior fragment lies external to the anterior, the inward and upward movement is prevented by contact with the anterior fragment; in other words, the movement of the fragment is controlled.

Fractures of the ramus are generally accompanied by much swelling, but usually the displacement is slight. With transverse fractures, the upper fragment, to which the temporal muscle is attached, moves a little forward, while the lower fragment moves a little backwards and inwards.

In *fractures of the neck of the condyle* the mandible is drawn upwards on the injured side and also over to that side, the condyle being drawn forward on the eminentia articularis by the external pterygoid muscle. The distal molar teeth on the injured side are in contact, the remaining teeth being separated. This malocclusion is brought about as follows: The upward pull of the muscles on the injured side brings the external aspect of the outer cusps of the mandibular teeth hard against the inner aspect of the outer cusps of the maxillary teeth; a continuation of the upward movement tends to rotate the jaw around these points of contact, as shown in the diagram (fig. 904).

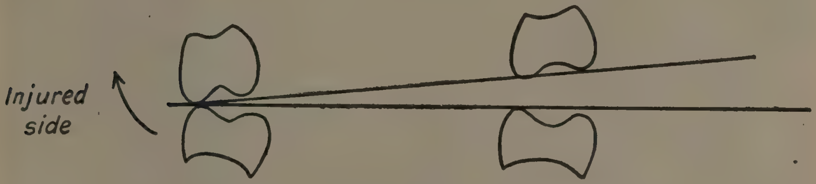

FIG. 904. (From *Brit. Dent. Journ.*)

When both condyles are fractured, the mouth is 'gagged' open on the posterior teeth, producing a condition of 'open bite.'

When the *coronoid process* is fractured, there is no displacement with the teeth in occlusion, but, on opening the mouth, the mandible may deviate away from the injured side owing to the unopposed action of the temporal muscle on the uninjured side.

A severe blow on the chin may fracture the mandible in the incisor region on the one side and through the ramus on the other side. A typical instance is shown in figs. 905 and 906. On the left side the bone was fractured in the position of the

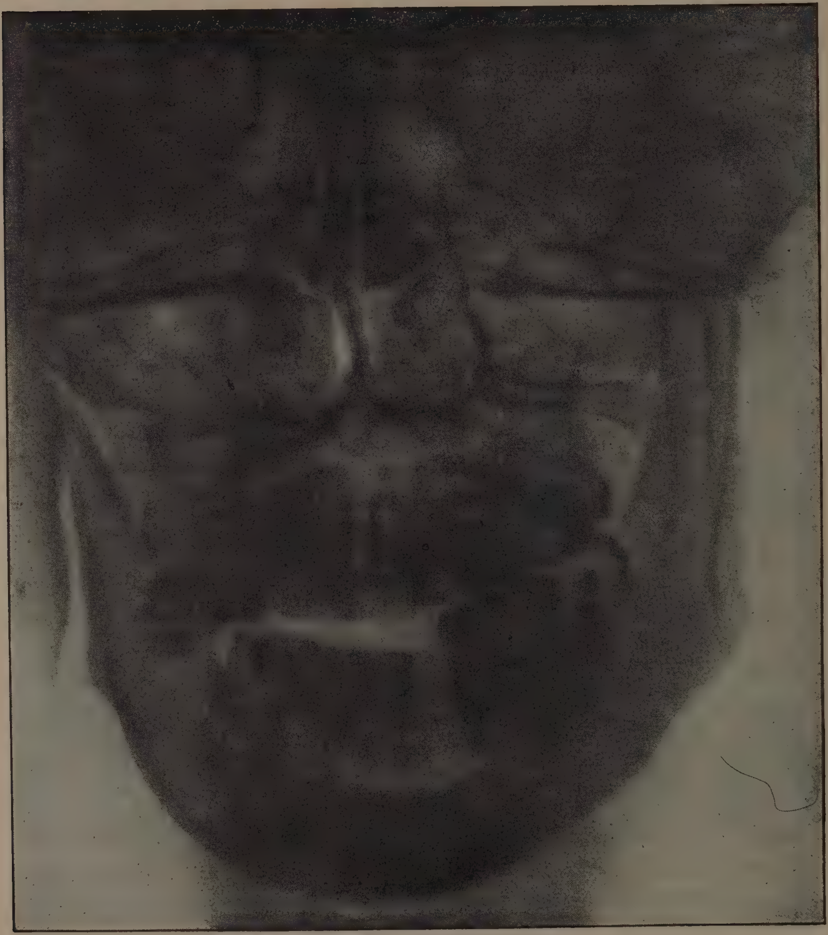

FIG. 905. (From *Brit. Dent. Journ.*)

canine in an oblique direction, and on the right side the ramus was split in a vertical direction. The displacement in this type of injury is as follows: The left fragment passes to the right; the central fragment is rotated so that the anterior end is displaced downwards and the posterior end upwards. The depressors of the jaw being attached to the anterior end, the pull of each set of muscles is in opposed directions, and the fragment is rotated at the area of contact between the last molars.

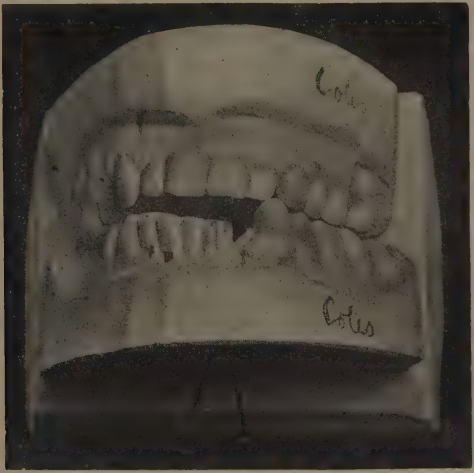

FIG. 906. (From *Brit. Dent. Journ.*)

With severe injuries, such as those caused by a kick from a horse, the jaw is often fractured in several directions, the force and direction of the blow being a potent factor in determining the degree of displacement. As an example the case shown in figs. 907 and 908 may be quoted. The injury was due to a mule kick in the region of the first molar on the left side.

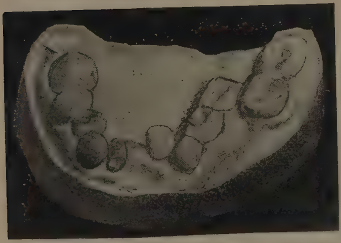

FIG. 907.

(From *Brit. Dent. Journ.*)

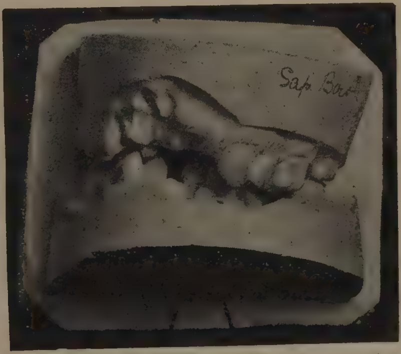

FIG. 908. (From *Brit. Dent. Journ.*)

The bone injuries were: (i) A fracture near the mid-line running downwards and backwards to the lower border of the bone; (ii) a vertical fracture between the first and second molars; (iii) a horizontal fracture connecting the two vertical fractures (see fig. 909). There was practically no comminution of the bone. In this case the left lateral fragment had swung forwards and upwards, and, owing to the backward displacement of the central fragment, the first molar was lying on the inner side of the second molar. The right lateral fragment had swung over to the left and was slightly depressed.

(ε) *Complications.*—The principal immediate complications in fractures of the mandible are extensive bruising of the soft tissues with, in a few cases, considerable laceration. Hæmorrhage is in some cases troublesome and seems to occur most frequently in fractures about the angles. It is probably due to the artery being torn and not completely divided. In rare instances a traumatic aneurism forms, and necessitates the ligature of the external carotid. The fracture may be associated with a dislocation of the temporo-mandibular articulation and occasionally with a fracture of the base of the skull.

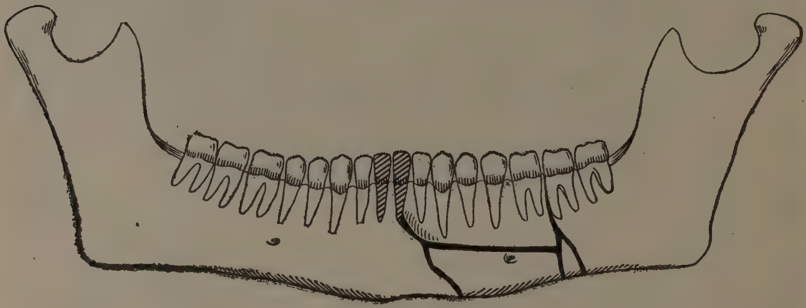

Fig. 909. (From *Brit. Dent. Journ.*)

(ζ) *The Healing of Fractures of the Mandible.*—Repair of the fractured mandible takes place in a manner similar to repair of long bones. Briefly, the stages are as follows:—

(i) The fractured ends are surrounded with a coagulated mass of leucocytes, blood and exudation from the vessels in the neighbourhood.

(ii) The coagulum is invaded by cells—polynuclear from the surrounding soft tissues, fibroblastic from the periosteum and medulla.

(iii) The coagulum is in time replaced by tissue derived from the periosteum and medulla. This tissue is

(iv) Converted into cartilage. (In cases where the callus is small this stage may be lacking.)

(v) The cartilaginous tissue is converted into osteoid callus, *i.e.* the cartilage cells are modified into bone corpuscles with a deposit of calcareous salts in the matrix leading to the formation of imperfect bone.

(vi) The imperfect bone is in time replaced by lamellar bone.

In *simple fractures osseous union is usually complete in six weeks*, but in cases where the treatment has been delayed, or there has been difficulty in reducing the fragments, union may not be complete in less than eight or ten weeks. In cases of doubt a radiogram should be obtained.

(η) *Treatment.*—The treatment of fractures of the mandible consists in (i) control of the sepsis; (ii) reduction of the displacements; (iii) immobilisation of the fragments.

As soon as the patient comes under treatment the mouth should be frequently irrigated with a mild antiseptic such as 1 in 100 carbolic acid, and the gums swabbed once a day with a 2 per cent. solution of iodine in alcohol, and the fracture rested. The older methods of the skull and chin support, and the four-tailed bandage, are now practically discarded for this purpose as they exert too much backward pull on the mandible and are apt even in relatively simple fractures to increase the displacement; indeed, if there is much displacement, or portions of bone are missing, it is all-important to guard against any backward displacement and particularly of the falling back of the tongue, which may otherwise cause asphyxiation, and for this purpose support directing its chief pull on the mandible in an upward and forward direction must be used.

The simplest and best way of applying this is by making several turns of a calico bandage about 2½ inches wide around the head and fastening it off with a safety pin; another piece of the bandage about 18 inches long is then passed under the mandible just in front of the angle and avoiding the chin, and the ends fastened off with a safety pin on either side to the bandage already round the head. These safety pins should be placed as far forward on each side as each individual case allows, bearing in mind that the object in view is not only support but avoidance of any backward pull; should there be any tendency on the part of the head bandage to slip downwards, as is conceivable with some shaped heads, another strip of bandage may be safety-pinned across the top of the head to prevent this. The supporting strip under the mandible may be padded or not as occasion suggests (fig. 910). In practice this method has been found more satisfactory than the ‘barrel’ bandage, as it is simpler, easier to apply, not so liable to slip and a more forward pull may be attained.

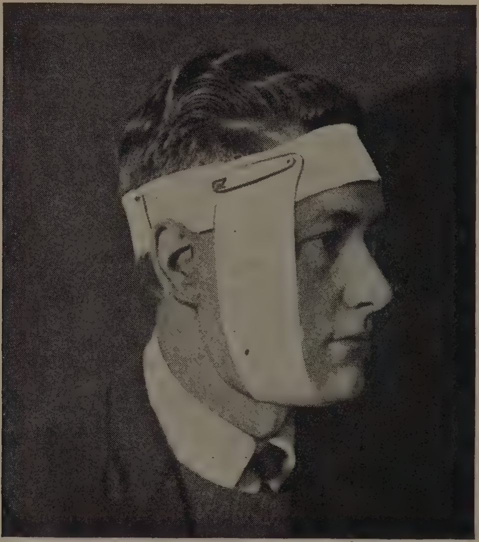

FIG. 910.

Radiograms should be obtained, and as soon as his condition permits the patient should be given an anæsthetic, in order that the teeth bordering on the line of fracture or communicating with the fracture may be removed as well as any septic teeth which may be present. If the extractions are likely to be difficult, nitrous oxide may be followed by the administration of ether. Before the operation is commenced the tongue should be brought under control by suitable means, and, during the operation, care should be taken to control the hæmorrhage.

The removal of the teeth in the region of the fracture is considered by some practitioners to be an unnecessary step, but in our opinion it is essential treatment, except possibly in very clean mouths. When the line of fracture passes through the socket of a tooth the periosteum of the root is detached and a pocket is formed which becomes filled with septic matter. The periosteum covering that part of the root is destroyed, and a permanent 'pocket' is formed from which infection is constantly passing to the fractured area. The removal of the tooth or teeth eradicates this focus of infection and rapid healing follows. A perusal of the records of the cases quoted in the following pages will convey an idea of the results obtained by free extraction of the teeth in the region of fracture. But the best evidence in favour of this method of treatment is to be found in a study of cases of delayed union. The radiograms of many of these cases show the roots of teeth in the line of fracture, and the removal of the teeth is invariably followed by rapid healing of the fracture.

The *immobilisation of the fragments* may be carried out in several ways.

(i) *Wiring.*—The operation of immobilising the reduced fragments of a fractured mandible with the teeth in occlusion by means of interdental wiring is sometimes used, chiefly as a temporary measure pending permanent immobilisation by means of cemented splints, but it is also advocated by some as a permanent means of treatment, though these latter are comparatively few.

Until recently any form of wire used for orthodontic purposes has been used, but there is no doubt that soft tempered stainless steel wire of not less than 0·4 mm. thickness is much to be preferred on account of its great tensile strength relative to its gauge.[1] On manipulation soft tempered stainless steel wire rather rapidly loses its softness, and is apt to become more springy.

The method of wiring which appears to have most advocates is that known as **'eyelet' wiring,** and is carried out as follows :—

A piece of wire about 8 inches long is bent double, and at the bend any circular instrument of about $\frac{1}{16}$ inch diameter is interposed, and turned round so as to twist the two wires together for three or four turns close up to the interposed instrument, thus forming a small eyelet at that point ; the free ends of the wire are then passed through an interdental space from the buccal aspect at a point where it is desired to fix the mandibular to the maxillary teeth in occlusion ; the free ends are then turned apart and passed singly and respectively from the lingual aspect through the interdental spaces immediately anterior and posterior to the space through which the double wire was passed so as to embrace the two adjacent teeth ; the end of the posterior wire is

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[1] Such wire is made by Messrs. Temco, Ltd., Lydbrook, Gloucestershire.

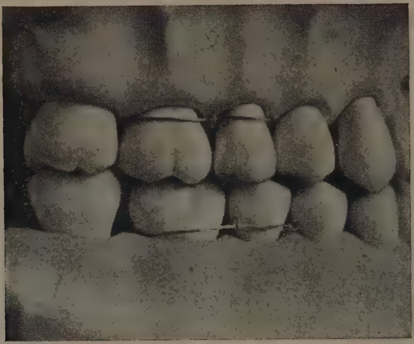

FIG. 911. (D. C. Simpson.)

then passed forward through the eyelet and the wire then taughtened and the two ends twisted together, cut off, and turned in (fig. 911). A similar adjustment is made in the opposite jaw so as to leave the maxillary and mandibular eyelets in such

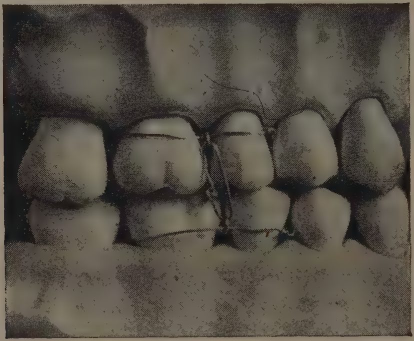

FIG. 912. (D. C. Simpson.)

position that when they are wired together the teeth will be held in their normal occlusion (fig. 912). It is obviously best to limit the number of eyelet wires to the requisite minimum to maintain stability and relative immobility in any given case, and the wiring may be tightened subsequently should this become necessary, as it frequently does.

The *advantages* of this method of treatment are that early and sometimes immediate treatment may be given, and the result attained judged to be so good as to be allowed to remain as the permanent treatment; the bite is not raised and if no anæsthetic has been needed so much the better. The *disadvantages* are that an anæsthetic may be required, the jaws are fixed and so the teeth not readily kept clean, there is difficulty in feeding in a moderately full-toothed patient, it is not easy to control sepsis and its possible sequelæ, wiring is not easily done and takes some time to apply, wires frequently become loosened and may thereby pull off the ligatured teeth, wire other than stainless steel is apt to break, and it is not usual for absolute immobility to be attained; there is too the risk that fractured jaws are not infrequently associated with cranial injuries, and such injuries may give rise to increased intra-cranial pressure and so produce a predisposition to vomiting, and vomiting with the jaws wired together may lead to disastrous results; obviously, if any possibility of vomiting exists when this form of wiring is used the nurse in charge of the patient must be provided with wire-cutting pliers and know which wires to cut, so that this may be done immediately.

Complete immobilisation by means of cemented splints appears therefore to be preferable as a means of permanent treatment.

Another form of immobilisation by means of wiring is by the use of **Labial Arch Wires**. These are wires which may be bent to fit round the labial aspect of the teeth of any arch, and are fixed by being wired to the teeth; each arch has a number of hooks or studs soldered to its labial aspect, so that by means of wire or elastic ligatures traction or fixation may be made from the maxillary to the mandibular arch.

These arches may be used with few or many teeth present; they are clean and accessible, and the direction of traction may be altered as occasion demands by affixing the traction wires or elastics to hooks or studs which are not opposite each other; if necessary, owing to displacement of the mandibular fragments, the mandibular arch may be made in more than one piece, or several pieces, so that the displacement may be gradually reduced.

(ii) *The Metal Cap Splint.*—This splint consists of a metal cap fitted accurately to the teeth and is fixed into position by means of oxyphosphate of copper cement. Double metal cap splints embracing both maxillary and mandibular teeth are

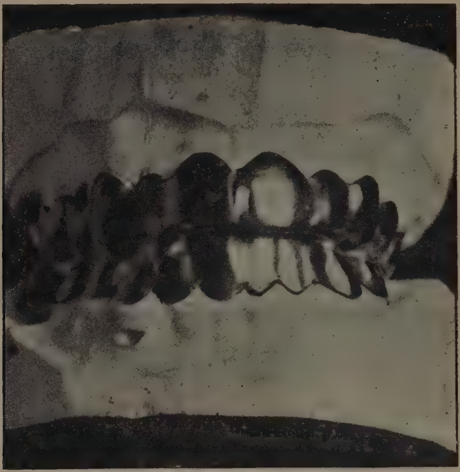

FIG. 914.

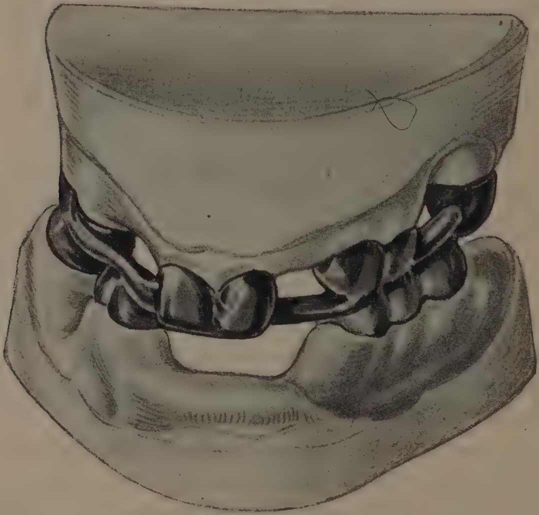

FIG. 915

the most reliable of all the types of splints (see figs. 914 and 915).

For the construction and adaptation of this type of splint the procedure is as follows :—

(*a*) Accurate impressions of the teeth are obtained.

(*b*) These are cast in plaster and the mandibular model divided at the point of fracture ; the two parts of the mandibular model are then placed so as to bring the mandibular teeth into correct occlusion with the maxillary teeth and both are mounted in this position on an articulator.

(*c*) The splints are then modelled in wax (No. 6 or 8 thickness), the wax being carefully pressed between the teeth and extended as far as the gum margin.

(*d*) At this point it is well to press the softened wax occlusal surfaces together, so that the bite will not be unduly opened when the splints are fixed and that greater perfection of occlusion may be obtained.

(*e*) The splints are then cast in Victoria metal or a copper-silver alloy and studs or loops soldered to the buccal aspects so that when in position they may be wired together.

(*f*) The splints are then adjusted in the mouth, the fracture reduced, and the splints fixed in position with oxyphosphate of copper cement, the maxilla and mandible being then wired together to ensure immobility of the fragments.

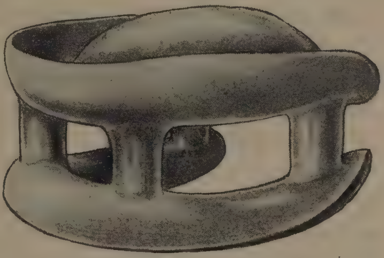

Fig. 916.

If the fracture cannot be readily reduced it may be necessary to make the splint for the mandible in two parts. This alternative method has been practised for many years, and in the majority of cases where there is displacement or missing tissue is preferable if not a necessity.

If there is but little displacement, when the position of each splint has been exactly localised they are removed and joined by a stout soldered bar, and the splint then cemented into place ; but if there is much displacement this is reduced after the cementing of the splints into place, and may be done (a) *Immediately*, by the use of wire ligatures uniting the maxillary and mandibular splints, (b) *Gradually*, by elastic force, or (c) *Manually* under an anæsthetic.

When there has been much loss of tissue between the two fragments, small splint-metal blocks should be attached to the labial aspects of the anterior portion of each of the mandibular splints, and these should be drilled and tapped so that after reduction a connecting bar may be screwed into place so as to make the whole into one rigid splint.

In these cases it is often advisable to take the impressions for the two parts of the mandibular splint separately.

Occasionally in fractures of the mandible when there is not much displacement a *single metal cap splint* may be used.

In many cases where metal cap splints are used the occlusal aspects may be cut away so as to produce *open cap splints*, and so restore natural occlusion and bite, and greatly increase the comfort of the wearer.

(iii) *The Gunning splint* (fig. 916) consists of vulcanite caps joined by supports, and constructed to fit both the maxillary and the mandibular teeth or gums. When the splint is in position a skull and mandibular splint or a four-tailed bandage is applied. With this type of splint the fractured jaw is fixed between the mandibular splint outside and the cranium. The Gunning splint is useful in cases where the patient is edentulous or possesses few teeth.

In many cases a combination of metal caps and vulcanite is found very useful. There are many other types of splint, but the experience gained from the treatment of war injuries has shown that the best results are obtained with either the metal cap splint or the Gunning splint.

In treatment there is always a difficulty in controlling the posterior fragment, and in some cases the only available fixed point is the third molar, whose stability is not always to be relied upon owing to the shape of its roots. It has also been observed that, if the posterior fragment overlaps the anterior fragment, union generally follows, the anterior fragment acting as a resistance against the inward pull of the superior constrictor and mylohyoid on the posterior fragment.

(iv) *Wiring of the Fragments.*—In a few cases where the fragments cannot be controlled by means of interdental splints, immediate wiring of the fragments may be adopted, but unless this can be made an extra oral operation it is not very successful, as infection is practically always present in fractures of the horizontal ramus.

Another method of wiring is occasionally used, principally for oblique fractures in edentulous mandibles when there is much displacement which is not readily reduced. This method is known as **circumferential wiring,** and consists of the forcible apposition of the fractured aspects of the bone by means of a wire entirely surrounding the mandible. The wire may be applied as follows :

A shallow vulcanite splint is first made to cover the alveolar ridge. Under anæsthesia a small incision is made in the skin along or slightly internal to the lower border of the mandible at the point at which it is desired to apply the wire, a fine trocar and canula (the latter without a shoulder) or a hollow stainless needle in a detachable holder (canula or needle to be about 15 Birmingham wire gauge), is inserted at this point and directed obliquely upwards close to the buccal aspect of the mandible till it emerges into the buccal sulcus close to the mandible ; the wire is then passed along the needle or canula into the mouth and the canula withdrawn ; it is then reinserted in the same incision alongside the wire already passed till it emerges on the lingual aspect of the mandible opposite the point of emergence on the buccal aspect, the other end of the wire passed up through the canula and the canula or needle withdrawn through the mouth.

The mandible is thus surrounded by a wire which encloses the fractured portions of bone, and with the splint *in situ*, and while the displaced or overlapping portions of bone are manually reduced as far as possible the wire is taughtened and twisted together over the top of the splint so as to hold the parts in apposition ; the ends are then cut short and turned over and a dressing applied to the small external wound.

Some pain and occasionally considerable swelling are apt to occur, and complete reduction and fixation are not necessarily attained by this means. The procedure is not often used and generally not much favoured ; besides, it is a fact that though of course ideal reduction should be one's aim, a slight degree of displacement in a fractured edentulous mandible is not a serious disability so long as bony union does take place, as any resulting deformity of the alveolar ridge can always be rectified afterwards in the construction of the denture.

(θ) *Delayed Union.*—Uncomplicated fractures of the mandible if properly treated should repair within three months, and if at the end of that period union has not occurred the case must be regarded as one of 'delayed union.' When there is delayed union there are three possible issues, namely :—

(i) Osseous union.

(ii) Union by a band of dense fibrous tissue, the fracture having a slight 'give.'

(iii) Non-union, the fragments being freely movable.

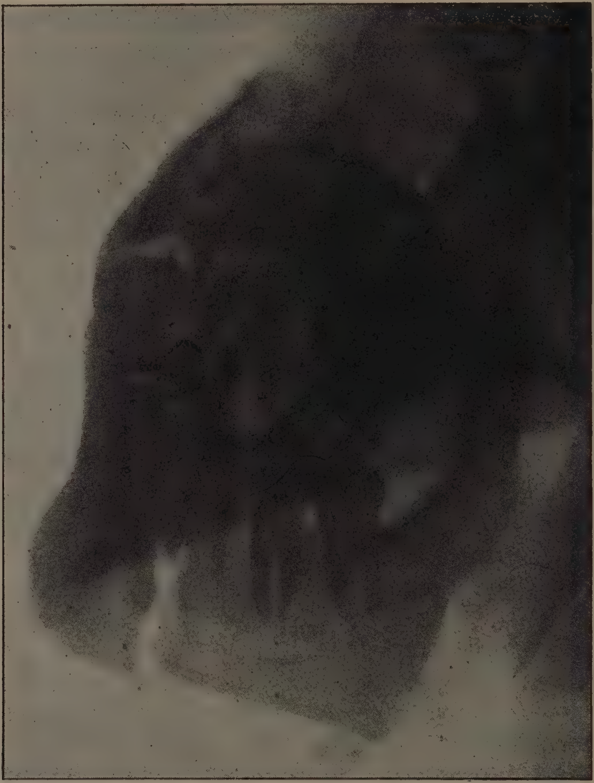

FIG. 918.—Radiogram of a case of 'delayed union' showing the root of a tooth in the line of fracture. (From *Brit. Dent. Journ.*)

The commonest cause of delayed union is the presence of septic teeth in the line of fracture. An example is shown in fig. 918. The radiogram was taken three months after injury. The removal of the affected teeth was followed by rapid union.

Occasionally the infection from the teeth leads to extensive rarefaction of the bone, or, if the infection is mild in type, to a sclerosis of the margin of the fragments. Both of these conditions result in non-union.

Another cause of delayed union is the presence of a thin layer of necrosed bone between the fragments. Radiograms of recent fractures sometimes show a thin detached portion of bone between the fragments. This thin layer of bone may become infected and necrosed and so lead to non-union.

Imperfect reduction of fractures leaving considerable overlapping of the fragments leads to prolonged delay, and so also does non-immobilisation of the fragments. The latter condition cannot always be prevented where teeth are missing. With fractures posterior to the last molar tooth the constant movement of the fragment by the superior constrictor of the pharynx is a common cause of delayed union or even non-union.

Even where the fragments have been immobilised and danger from sepsis has been quickly eliminated union is at times delayed. This is probably due to defective power of repair on the part of the individual, possibly dietary in origin.

With diabetics, or where there is a history of syphilis, union is at times delayed.

In cases of non-union, sepsis, if present, should be removed, and when the tissues have thoroughly recovered, the ends of the bone should be freshened sufficiently to expose the medulla and the fragments approximated and immobilised either by means of interdental splints or by an extra-oral wire suture through the bone.

When a larger portion of bone is missing, as may occur when some necrosis has taken place so that approximation of the two fragments would produce deformity or malocclusion, the two portions of mandible may be splinted into correct occlusion with the maxillary teeth and retained there till healing is completed; a bone graft, usually a portion of iliac crest, may then be inserted and the splints retained till bony union has taken place; there is a small risk of latent sepsis interfering with the success of the operation.

The progress of union may be watched by means of periodic radiograms, but callus is not always easy to see in radiograms, even when a soft tube is used. The bone graft operation should, of course, be an extra oral one, and comes into the domain of the general surgeon, working usually—and advisedly—in conjunction with the dental surgeon.

(ι) *Mal-union.*—Where the occlusion is only slightly deranged the simple operation of 'cutting in the bite' will often suffice to correct the malocclusion. In the case of fractures in the region of the last molar tooth and fractures of the ramus the mouth may be 'gagged' on the posterior teeth, and these teeth will need to be removed in order to bring the remaining teeth into occlusion. Failure to reduce the fragments correctly may result in marked deformity. For example, in fractures behind the last standing tooth, the posterior fragment may swing forward and the covering muco-periosteum come into contact with the maxillary teeth. Discomfort and perhaps ulceration of the soft tissues follow. The patient, in order to avoid the discomfort, involuntarily brings the jaw over to the injured side and the face becomes markedly deformed. In other words, the patient acquires a 'bite of comfort' at the expense of his personal appearance. A similar condition is seen in fractures of the molar region where the anterior fragment has moved to the injured side and union has followed. An attempt to bring the mandible over to the uninjured side is resisted because the soft tissues covering the ramus would then come into contact with the maxillary teeth.

The treatment consists in the removal of one or more of the maxillary molars on the injured side, the patient being encouraged to use the teeth on the uninjured side. In some cases the upward movement of an edentulous posterior fragment is so extreme that the fragment lies external to the alveolar process of the maxilla. In such cases extraction of the teeth is not always sufficient to bring about union, and it is necessary to cut away the bone freely, including the tuberosity. If the mal-union is of long standing, it may be necessary to train the mandible into position, either by means of an engaging flange, or by means of stretching splints. An engaging flange is an apparatus adjusted to the teeth in such a manner as to cause the teeth to occlude in a normal position.

The stretching splint is made as follows :—

(i) Metal caps are adjusted to the maxillary and the mandibular teeth.

(ii) The maxillary splint is covered with a layer of soft composition. A grip of the mandibular teeth on the injured side is obtained and, as the mouth closes, the mandible is firmly brought over to the uninjured side.

(iii) The splints are soldered together ; the upper cap is placed in position and the mandible guided into the lower splint.

(iv) The mandible is retained in this position until the patient can easily bring the teeth in and out of the splint.

(v) The splints are unsoldered and the mandible is brought over still further to the uninjured side, the operation being repeated until the correct position is obtained.

Changes in the nerves of teeth anterior to the line of fracture have been found by R. V. Bradlaw,[1] who describes and figures marked fragmentation and sometimes vacuolation in the pulpal nerves of these teeth.

(2) FRACTURES OF THE MAXILLA.

(α) *Causes and Position.*—Fractures of the maxilla are generally caused by great violence. This type of fracture not infrequently results from airplane accidents. When the machine strikes the ground, the pilot's head is jerked forward and his face comes into contact with the lower part of the frame of the observing screen. Frequently the bone is comminuted ; adjacent bones, such as the malar and nasal, are implicated, and the base of the skull may be fractured. When the impacting force strikes the bridge of the nose in a downward direction, as in lift accidents, a transverse fracture of both maxillæ may result, the line of fracture passing high up and involving the infra-orbital plate. In such cases the whole maxilla can be moved *en masse*, and there is extreme ecchymosis of the soft tissues. In some cases the bone is forced downwards and backwards and becomes impacted, the bite being 'gagged' on the molar teeth. If the impact is received on the lower part of the maxilla, multiple fracture of the lower portion of the bone results.

Separation of the two halves of the maxilla in the median line has been recorded. Lastly, *fracture of portions of the alveolar process and of the tuberosity* occasionally occurs during the extraction of teeth.

(β) *Complications.*—These are similar to those met with in fracture of the mandible. The swelling of the soft tissues is, however, often more severe, and there is a greater liability to swelling of the tissues of the naso-pharynx and larynx. The swelling around the air passages must be carefully watched and if necessary laryngotomy performed. Hæmorrhage is at times severe, and it is important to avoid the use of peroxide of hydrogen as the frothing produced may cause obstruction of the air passages.

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[1] *Proc. Roy. Soc. Med.*, 1939, xxxii, 1040.

(γ) *Method of Union.*—Repair in the fractured maxilla appears to be invariably of a fibrous and not of an osseous character. One has examined many cases and been unable to satisfy oneself that osseous union has actually occurred in any of them.

(δ) *Treatment.*—In **transverse fractures above the level of the roots of the teeth** the maxilla may be immobilised by means of a metal cap splint cemented to the maxillary teeth ; to the sides of the splint stout wire bars are soldered which come out at the sides of the mouth and curve backwards so as to lie close to the cheeks ; these in turn are attached to vertical metal struts, the ends of which are fixed in a plaster head cap.

There are various ways of completing this fixation and of making a plaster head cap. That used and described by H. M. Crombie[1] is practical and simple ; he describes it as follows :

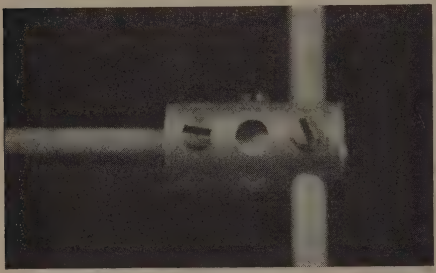

Fig. 919.—Coupling connecting a vertical strut to a bar extending from the side of the splint. Slightly enlarged ; actual length of the coupling is about $\frac{13}{16}$ inch.

'The head gear is a plaster head cap in which are embedded rigid struts rigidly connected to the intra-oral apparatus. (Fig. 921.)

'The struts are ordinary Meccano axles or paling wire. Paling wire may be obtained of the same gauge as Meccano axles and is considerably cheaper, and has the advantage of being easy to obtain and simple to adapt to the requirements of the case. The struts are joined to bars from the intra-oral apparatus by means of three-hole Meccano couplings (Part No. 63) which can be adjusted to any angle and make for easy and rapid connection between them. The bars must be of the same gauge as the struts. (Fig. 919.)

'Taking as example a case in which the facial bones have been detached from the base of the skull and have dropped as a result of gravity : as a rule, it is easy to replace these in their proper position ; what is required is to hold them there until healing takes place. First make a metal cap splint to fit the maxillary teeth. On the sides of this splint are soldered bars, which should be gilded. These come out at the sides of the mouth and curve backwards to lie close to the cheeks. The struts which are to be embedded in the head cap are now bent at the upper end to follow the curve of the skull. They need not extend to the vertex, in fact it is better if they do not. A coupling is put on the upper end of each strut by its middle hole so that it forms a " T " shape to give adequate retention in the plaster. The struts are then put through the holes in couplings attached to the bars on the splint and adjusted to lie close to, but not touching, the skull. The splint is then cemented on to the teeth.

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[1] *Brit. Dent. Journ.*, 1941, lxxi, 297.

'The patient's hair having been cut, a stockinette cap is put on his head. It is not necessary to introduce any padding; indeed it is better to do without it as it tends to make the cap less secure. A plaster bandage is then wound round the head; about six turns is sufficient. The struts are then put in close contact with the plaster, and short lengths of plaster bandage packed in under and along the sides of them so that there is no gap between them and the plaster bandage already applied. When the struts are buttressed in this way, another plaster bandage is wound round the head over the top of the buttressed struts and smoothed into place as one proceeds. It is not necessary, as a rule, to put on more than another nine or ten turns, the number depending on the amount of strain the plaster may have to withstand, and the lighter the skull cap can be made the better. Finally, some plaster is mixed of the same consistency as used to cast a model and smeared on so that the network of the bandage is obscured. Then, with a wet mop, the whole is smoothed. This prevents the cap becoming dirty, as it will if left with a matt surface. One then tightens the set screws in the couplings connecting the struts to the bars in the splint and has a rigid apparatus which will not move under stress. (Figs. 920 and 921.)

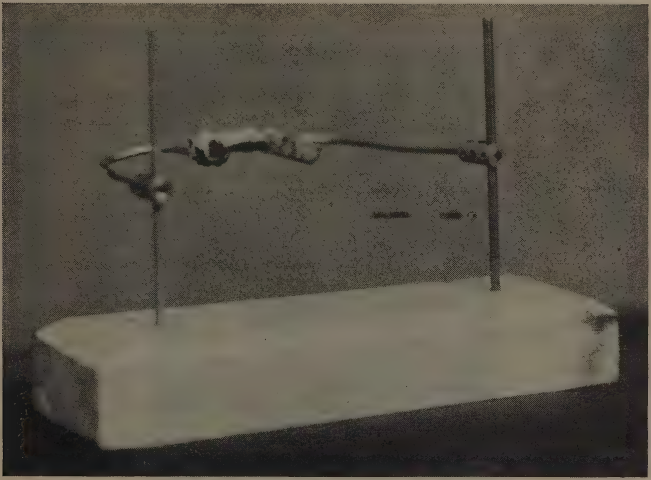

FIG. 920.—The apparatus obliquely from behind.

'Certain points must be watched in applying the plaster bandages. They should go well down the forehead, but about an inch should be left between the lower edge of the plaster and the eyebrows. They must be kept well down over the parietal eminences, must leave the ears clear and be kept well down over the occipital protuberance. Do not cover the crown of the head.

'For increased rigidity the two struts may be joined across the front of the face with a cross bar attached by couplings, but it is desirable to avoid interfering with the patient's vision.

'It is possible with this apparatus to exercise traction on the maxillary bones by loosening the set screws and moving the couplings with the splint up or down the struts, as the case may be, a little every day. One can also get antero-posterior movement by loosening the set screws on the arms of the splint and moving them a little forwards or backwards and refixing every day. By manipulating one of these or by moving them in opposite directions, one gets a certain amount of rotation in the horizontal plane.

'This apparatus will not only hold the face up, but will also hold it down, if necessary. It is simple to construct, can be used in conjunction with any type of metal cap splint, and is not heavy.

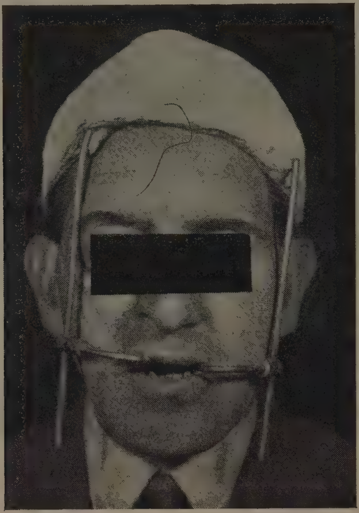

FIG. 921. (H. M. Crombie.) (From *Brit. Dent. Journ.*)

'The application of this method to an edentulous jaw presents no difficulties. If the patient has an upper denture, all that is necessary is to embed the curved bars in the sides of the denture, and vulcanise them in. If he has not an upper denture, or if it is damaged beyond repair, it is a simple matter to make one without teeth to fit the palate and embed the bars in that. There is no need to remove such a denture for cleaning, provided there is no actual wound of any size in the palate, as foreign material does not tend to get underneath and the mucous membrane is barely reddened when the apparatus is removed, often after several weeks.

'This method can also be applied to emergency splints. All that is required is a few box trays of different sizes with the handles cut off and curved bars soldered to the sides. In an emergency fill one of these with modelling composition, press it gently home, put on a plaster head cap, which can be done in five or ten minutes, and attach struts from the head cap to the bars by means of couplings. It would not be necessary to wait long for the plaster to set before joining up the struts to the bars since no great strain will be put on the cap with such an apparatus.'

In **multiple fractures through the alveolar portion of the bone** the teeth involved should be removed and a Gunning splint used until repair has taken place.

The removal of the teeth must be carried out with extreme care. It is often a useful plan to grasp the tooth next to the one to be removed with a pair of forceps and exert pressure in an upward direction during the extraction. If this precaution is not taken undue laceration of the parts may occur. At times the teeth are more easily removed by exerting force in a lateral direction with a straight elevator, and if the muco-periosteum is unduly adherent it should be pared off with suitable instruments.

When there is considerable comminution of the teeth the muco-periosteum should be raised from the outer aspect of the bone and a view obtained of the alveolar process ; the broken fragments can then easily be removed together with loose pieces or sharp edges of bone, the muco-periosteum being brought together with sutures.

In the maxilla the line of fracture may run immediately above the roots of the molars, the whole segment of bone being detached. Union under these conditions does not occur, and it is better to remove the whole segment. The muco-periosteum is first reflected from both sides of the bone and the detached portion carefully dissected out, the edges of the soft tissues being brought together with sutures.

The rapidity of the recovery of patients with severe multiple fracture of the maxilla, when the teeth are removed, is remarkable. Moreover, the teeth if retained are of no value for mastication, the bone around them is liable to necrose, and the maxillary sinus to become infected.

(B) Gunshot Injuries of the Jaws.

In times of peace, gunshot injuries of the jaws are of rare occurrence. As these injuries will in the future come more and more under the care of the dental surgeon it is necessary that he should possess some knowledge of their nature and the best methods of treatment.

In gunshot injuries of the jaws there is frequently considerable loss of tissue and much comminution of the bone, and in these respects they differ from the jaw injuries ordinarily met with in practice. The damage to the bone would seem in some measure to be governed by :—

(i) *The Type of Missile.*—A bullet tends to comminute the bone into smaller fragments than 'shrapnel.'

(ii) *The Velocity of the Missile.*—As a rule, the higher the velocity the greater the degree of comminution.

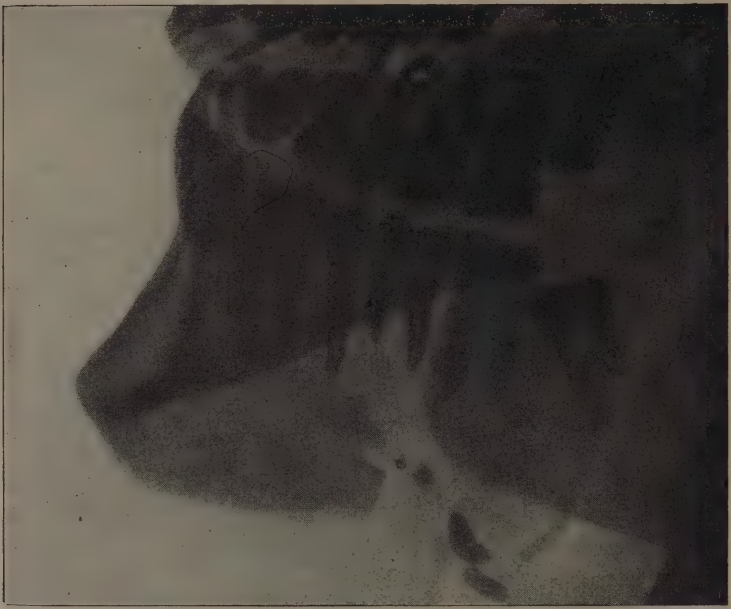

Fig. 922. (From *Brit. Dent. Journ.*)

(iii) *The Angle of Impact.*—A bullet which strikes the bone at a right angle cuts away the bone in its course, but does not necessarily cause extensive comminution of the remaining fragments. An example is shown in fig. 922. A bullet struck the bone at a right angle near the lower border and carried away the portion below the tooth. The tooth remained in position, being firmly wedged in place by the approximation of the fragments. If a bullet strikes the bone at an oblique angle widespread comminution is caused, but there is little loss of tissue. An example is shown in figs. 923 and 924. The bullet entered the lower lip one

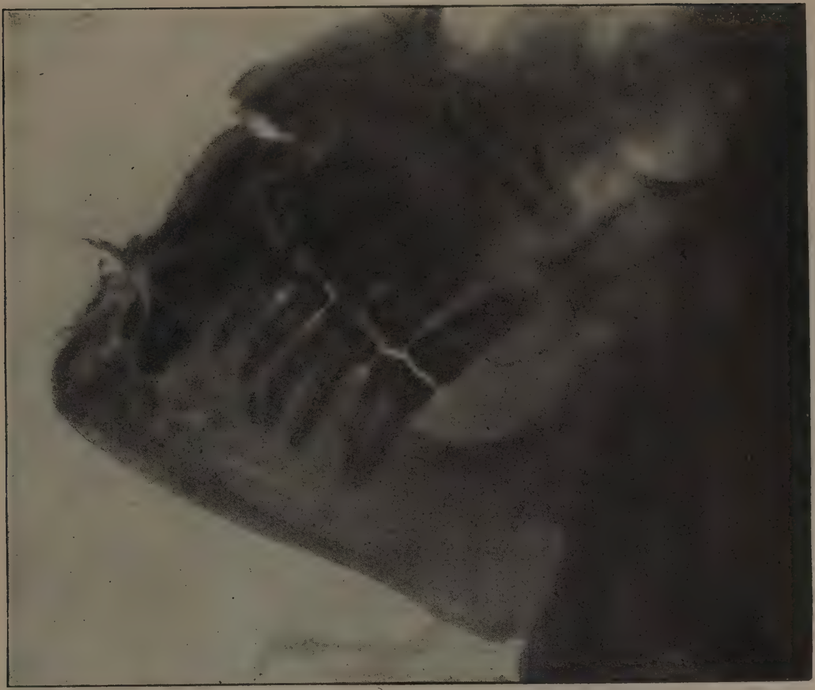

FIG. 923. (From *Brit. Dent. Journ.*)

inch to the right side of the mid-line and came out on the left side near the level of the hyoid bone at a point midway between the angle of the jaw and the symphysis. The mandible was therefore

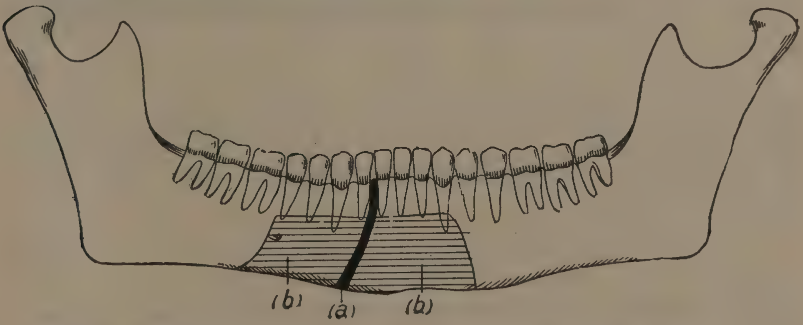

FIG. 924.—(*a*) Line of vertical fracture ; (*b*) area of comminution. (From *Brit. Dent. Journ.*)

hit at a very oblique angle, and the bullet took a slightly downward course. The bone was fractured in a vertical direction between the right first and second incisors, the comminution extending beneath the $\overline{54321|123}$.

The photograph of another patient shown in fig. 925 depicts the entrance and exit wounds, the former being on the cheek. It would seem from the direction taken by the bullet that the lower border of the mandible was little more than grazed, yet the concussion was sufficient to comminute the bone in the manner shown in fig. 926.

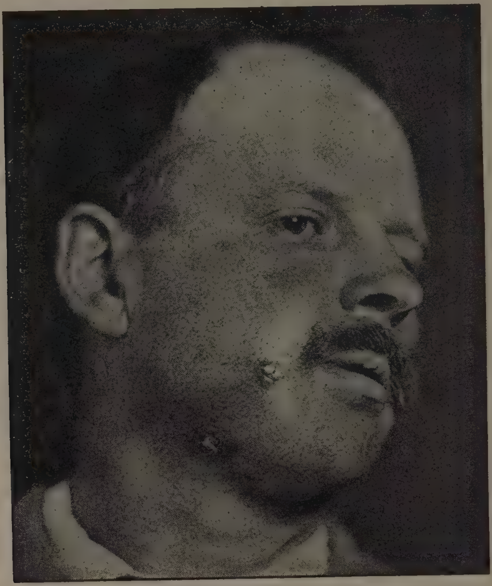

Fig. 925. (From *Brit. Dent. Journ.*)

(iv) *The Character of the Bone receiving the Impact.*—The harder the bone the greater the degree of comminution. A missile which strikes the hard compact tissue of the lower part of the body of the bone will cause more comminution than one which strikes the alveolar process.

Treatment.

(i) *General Lines of Treatment.*—The general lines of treatment to be adopted are briefly as follows: With much laceration and suppuration of the soft parts, the first object should be to clean the parts and remove the sepsis. For this purpose chloramine and eusol are very efficacious, and the rapidity with which wounds will clear up when treated with these preparations is very remarkable. The mouth is frequently irrigated with hypertonic saline, and the gums swabbed once a day with a 2 per cent. solution of iodine in alcohol. Radiograms of the injured area are next obtained, and here it should be mentioned again that both lateral and antero-posterior views are necessary. The next stage is to examine the patient under an anæsthetic. In cases of recent injury the examination has often to be delayed until the patient has sufficiently recovered from the shock of the injury to undergo anæsthetisation with safety. The anæsthetic should be administered with extreme care on account of the swollen condition of the soft parts, and the slightest interference with the passage of air to the lungs must be avoided. Before

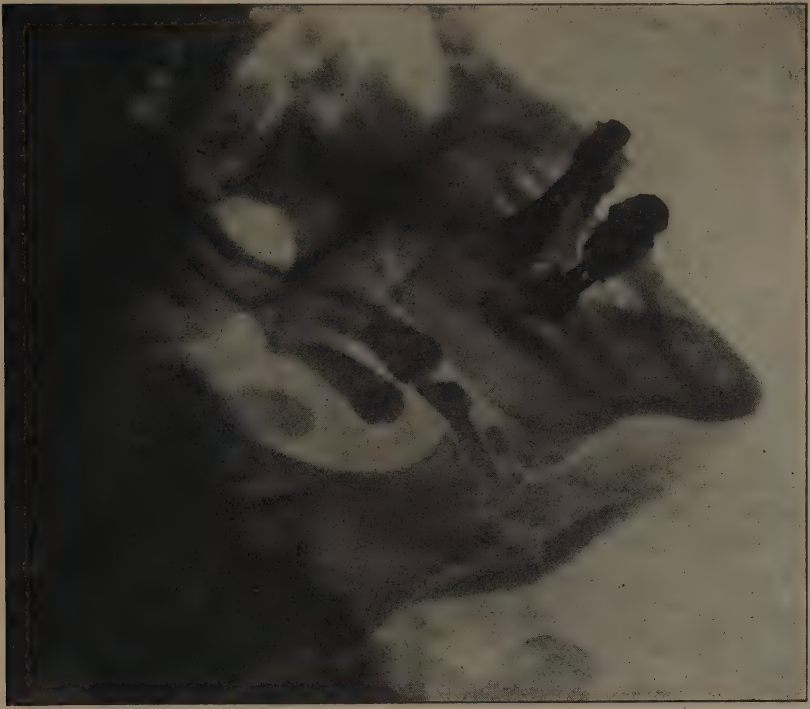

Fig. 926. (From *Brit. Dent. Journ.*)

the operation is commenced the tongue should be brought under control; this is especially necessary if the tongue is partially bound down by adhesions, or if a central fragment exists. The parts are next examined in order to ascertain how far reduction is possible. Teeth bordering on the line of fracture, or communicating with the fracture, are then removed, as well as any septic teeth. While the teeth are being removed, every precaution is taken to prevent blood passing into the pharynx. The extraction should be carried out very cautiously and with as little violence as possible, the teeth being, so to speak, gradually enticed from their sockets. Sharp edges of bone left after the removal of the teeth should be trimmed away and the edges of the muco-periosteum brought into apposition by sutures. The lacerated tissues of the cheek and lips should, if their condition permits, be brought together.

(ii) *The Approximation of the Fragments.*—To obtain osseous union it is essential that the fractured ends of the mandible should be brought into contact, or that living bone should exist in the tissues between the fragments of bone. The approximation of the fragments will result in a narrowing of the arch and some loss of masticating area, but the masticating area can be restored by properly constructed artificial dentures. Some writers who place considerable stress on correct occlusion of the teeth maintain that, if the fractured ends are kept apart, fresh bone will eventually bridge the gap. There is nothing in pathological teaching or experience to support this view, and if bone cells do not exist in the tissues uniting the fragments it is difficult to see how fresh bone is to be created. The cases in which bone has been observed to form have probably been examples of comminution of the tissues with the fragments of bone remaining and forming nuclei for bone growth. Experience shows that many of these cases end, as might be expected, in fibrous union. The choice would therefore seem to lie between a mandible firmly united and capable of bearing the strain of an efficient denture, or the teeth in correct occlusion on a base not capable of bearing the strain of mastication.

In the incisor region the fragments are allowed to approximate, provided that the narrowing does not lead to marked deformity of the face and disorganisation of the occlusion. An example may be quoted :—

The bullet carried away the bone, as shown in fig. 927. The parts were approximated, osseous union followed, and occlusion of the teeth was very slightly impaired (fig. 928).

In the molar region the following method is adopted: The maxillary teeth opposing the teeth of the posterior fragment are removed, allowing the posterior fragment to move forwards and upwards, and the anterior fragment to swing over to the injured side. Where the gap is small, the posterior fragment is released, and a splint made with the anterior fragment in position. Radiograms are then obtained. If the bony parts are found to be in contact, the splint is fixed in position; otherwise the anterior fragment is brought across until contact is obtained. The mandible may thus be mended with the anterior fragment swung over to the injured side, but this is no disadvantage because, with the removal of the splint, there is a tendency for the mandible to adjust itself to functional activity, provided that the so-called 'bite of comfort' is avoided (see p. 911).

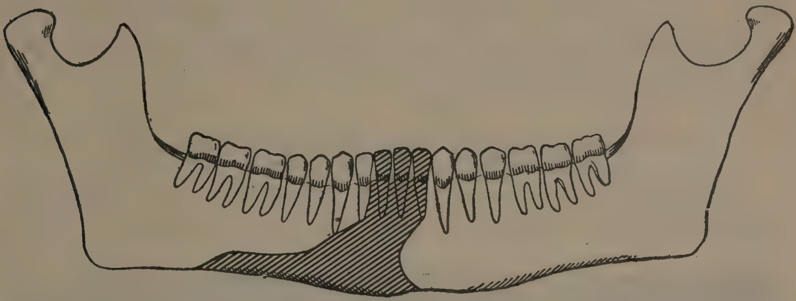
Fig. 927. (From *Brit. Dent. Journ.*)

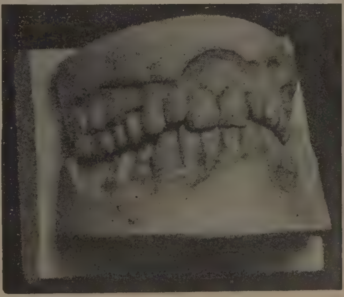
Fig. 928.
(From *Brit. Dent. Journ.*)

It is difficult to indicate to what extent the fragments may be allowed to approximate without causing undue deformity, and it is probable that the degree of approximation which is safely attainable differs in individual cases. Good results may be confidently anticipated if the gap is less than half an inch, and, even with gaps up to three-quarters of an inch, approximation may prove successful in some cases. With a wider gap, approximation of the fragments must give way to bone grafts for the formation of an osseous bridge.

(iii) *Fixation of the Fragments.*—Splints similar to those described on p. 905 *et seq.* should be used to fix the fragments. The splints should be adapted as soon as the condition of the tissues permits, but they should not be fixed until the sepsis is completely under control.

(C) Pathological ('Spontaneous') Fracture.[1]

'Spontaneous' fracture of the mandible is not under any circumstances common. It may occur if there is necrosis involving the whole depth of the bone, particularly if the necrosis is rapid; it may occur at the site of a neoplasm (fig. 929), and has been known to occur during the act of sneezing in an edentulous person having a history of chronic osteomyelitis of seven years' duration. It has also occurred when abscesses have been present and when the bone has been weakened by actino-

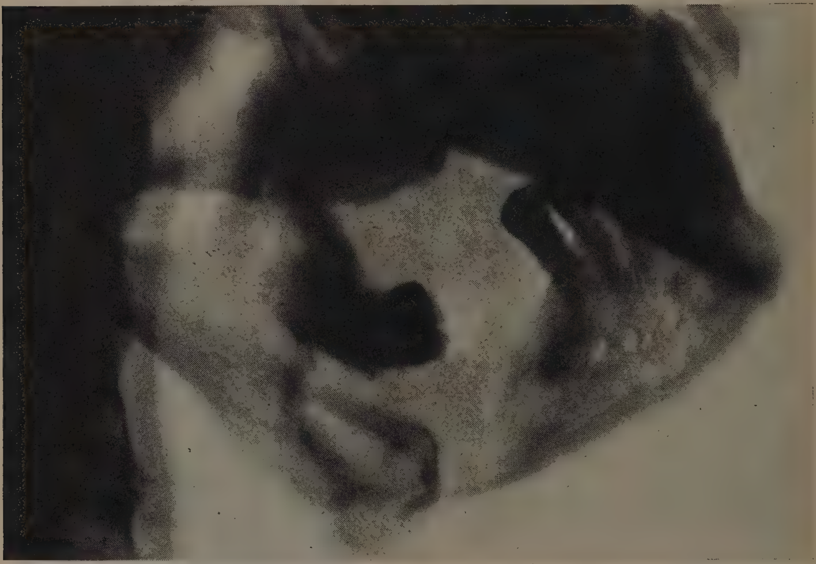

FIG. 929.—Pathological ('Spontaneous') fracture of right side of mandible in a male aged 62, resulting from carcinomatous invasion and erosion. (E. S.)

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[1] *Brit. Dent. Journ.*, 1935, 555. (E. S.)

mycosis. Indeed, any factor which tends to weaken or attenuate the bone may be a predisposing factor.

For this last reason *pathological fracture may also be a sequel to the extraction of teeth,* when infection extends via the socket through a vertical or oblique segment of the mandible causing its necrosis.

Usually such extension and necrosis is not particularly rapid, so that, by the time it has involved the depth of the bone and the necrosed portion separated, sufficient callus has formed to preserve continuity and prevent displacement. The callus thus acts as a natural splint till bony union is restored, when it is absorbed. If there is any necrosis, the wound does not heal till the last of the necrosed portion of the bone has come away, and this may be a long time in happening, even up to nine or ten months or more, though it usually takes place more rapidly. In such a case, three to four weeks after the extraction there is usually some, possibly considerable, pain, sepsis, discharge and swelling. This last is partly inflammatory and partly due to the formation of callus, and usually gives rise to some external asymmetry. So long as the necrosed portion of bone has not come away in its entirety these signs and symptoms will persist in greater or lesser degree ; there is, however, no undue mobility or loss of alignment in the bone, so that often no thought of the likelihood of fracture is seriously entertained.

If a radiogram be taken at this stage, appearances are apt to be disconcerting as, though callus may occasionally be faintly seen, there is usually no evidence of it, even in a good radiogram ; at the same time the necrosed segment of bone or the place that it once occupied is very obvious, so that the picture is that of a mandible in two pieces or nearly so. In other words, **there is a radiographic fracture,** and this appearance may continue for several months.

If, however, the infection is a little more virulent and/or the resistance of the patient so lowered that callus is not formed rapidly enough, in sufficient quantity or of efficient quality, the mandible may actually separate into two portions and a clinical pathological fracture occur. Still more likely is this to occur if it be exposed to even slight trauma at this period, such as would be negligible under normal circumstances. From the radiological appearances the risk of this happening seems to be at its maximum from about four to seven weeks after the extraction.

The following cases are illustrative of what may happen :—

In February 1934, a male, aged about 45, had an acute Vincent's infection, and though extractions are, if possible, avoided under these circumstances, it was found necessary to remove $\overline{87|}$, which were very loose. Infection of bone supervened, and ultimately a portion of bone, extending from $\overline{5|}$ to halfway up the ascending ramus on the same side and involving the whole depth of the jaw, necrosed, separated and came away four to five weeks later. Apparently the infection was so acute and rapid that there was not time or opportunity for the formation of an efficient callus support, and the jaw was in two separated portions, *i.e.* a pathological fracture. In this case it is quite possible that fracture would have occurred even if the teeth had not been removed.

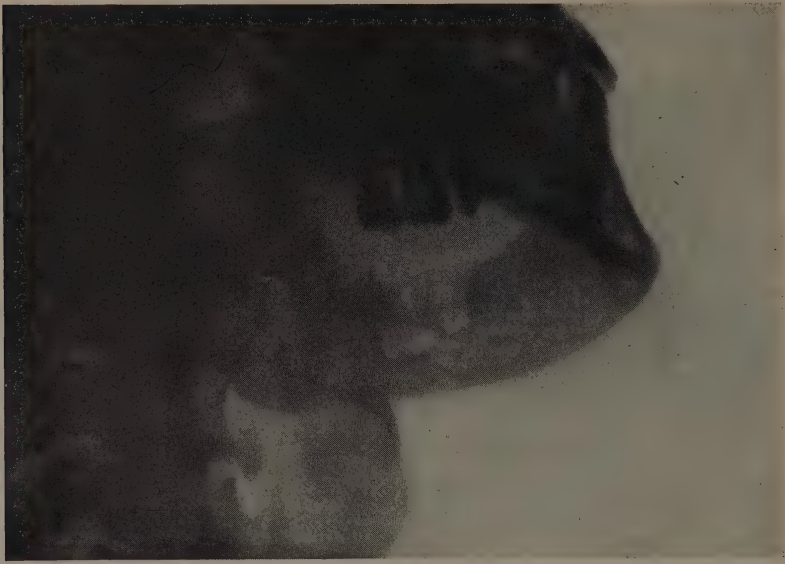

FIG. 930.—Forty-one days after initial operation, shows infection and necrosis extending almost through the depth of the mandible and a crack at the extreme lower border. (E. S.) (From *Brit. Dent. Journ.*)

On February 24, 1934, an attempt was made to remove a mandibular left third molar under local anæsthesia for a female aged 39; it was not entirely successful and a second attempt a few days later also under local anæsthesia had a similar result; at a third attempt a few days later also under local anæsthesia the tooth was removed. Fourteen days after the initial attempt there was much swelling of the cervical glands, the swelling spreading up over the mandible; there was also a discharge of offensive pus and a tendency to hæmorrhage, the temperature reaching to 103° F.; hot carbolic mouth washes were used internally and antiphlogistin applied externally; syringing was also resorted to. The wound became clean and the temperature fell under this treatment, several small sequestra came away and the cavity became filled with granulation tissue. Resolution seemed so well under way that a splint was not deemed necessary, particularly as it would have interfered with keeping the wound clean. Forty-one days after the initial operation there was a sudden attack of pain on opening the mouth and a radiogram (fig. 930) taken at this time, April 7, showed an area of infection and necrosis extending almost through the depth of the mandible in that region and a crack (fracture) at the extreme lower border; there is, however, no very obvious displacement. A large sequestrum, which subsequently came away, apparently aided in preventing undue displacement, but even so, muscular traction and the plasticity of such callus as was present did not prevent slight displacement ultimately occurring, and fig. 931, taken three and a half months after fig. 930, shows how small this was in amount.

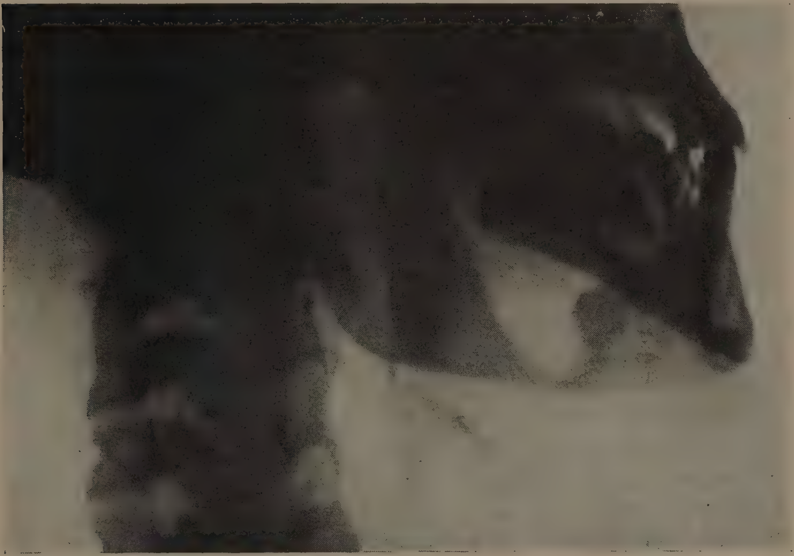

Fig. 931.—Nearly five months after initial operation, shows how small an amount of displacement ultimately resulted after this pathological fracture. (E. S.) (From *Brit. Dent. Journ.*)

A similar case in which fracture occurred following the extraction of an impacted mandibular third molar is recorded by H. J. Field in the *International Journal of Orthodontia*, 1929, xv, 1212. In this case pain was more pronounced forty-four days after the extraction, and at this time ‘ Roentgen studies showed further sequestration, making the division between the ramus and the body complete.’

These cases emphasise that, if any unusual force has been used in extraction, we should, for our own protection and our patient's satisfaction, have a radiogram of the site of operation after its completion, and (perhaps more important) that after any extraction when the wound does not show the usual signs of ready resolution, a radiogram should be taken about ten days after the operation, so that it may be seen if infection and necrosis are spreading, or have spread through the depth of the bone. If this is seen to be happening one may then take such precautions as seem necessary, even to the making of splints, with the hope that they will not be required, and the condition should be explained to the patient.

In the case of extractions where, for various reasons, difficulty is to be expected, it is usual to have radiograms before the operation is undertaken; these should be preserved, as they are sometimes evidence of a pathological condition of bone already present before operation. Needless to say, any subsequent radiograms should also be preserved. Radiograms for these purposes should be of sufficient size to show the whole depth of as well as the immediate bony environment of the tooth.

The usual term applied to fractures of this nature is 'spontaneous fracture.' 'Spontaneous' means 'self-initiated,' and that is true of no fracture. It is, of course, well recognised that the special technical meaning of this term is that it is a fracture caused by slight or trivial force, but in the type of fracture described separation might or would ultimately occur if no force had been used. The term 'pathological fracture' is better.

The importance of radiograms is well illustrated by the following case:

A patient with a very badly swollen face came to see a dental surgeon, stating he had been in that condition for some time; he had a severe alveolar abscess in connection with ⌈3, and ⌈45, one of which was broken down, also appeared to be involved. These three teeth were quite loose and easily removed under nitrous oxide anæsthesia. The patient did not attend again till 24 days later, when the mouth was still painful and the wound not healing, and there was now a sinus beneath the mandible and a loose sequestrum too large for removal through the extraction wound. A radiogram 31 days after the extractions showed an osteomyelitis and loss of bone involving the whole depth of the mandible in the area from which the teeth had been removed, *i.e.* a 'radiographic fracture' as described on page 925. The patient felt convinced that the mandible had been fractured during the extraction of the teeth, but the clinical and radiographic evidence taken together showed without doubt what had happened and how it had occurred.

CHAPTER XXX

Necrosis of the Jaws.

Causes—Signs and Symptoms—Diagnosis—Treatment.

Necrosis affecting the jaws may be limited to a small fragment of the alveolar process, or may involve the greater part of the bone. Necrosis is more common in the mandible than in the maxilla, on account of the exposed position of the former, its smaller blood supply, and the fact that it is composed mainly of compact tissue.

(A) CAUSES.

(*a*) **Infection.**—In the majority of cases, necrosis of the jaws is the result of septic or infective processes arising in connection with the teeth. The *infection may reach the bone via the teeth or their sockets*, and examples of this are met with in the necrosis which occasionally follows septic periodontitis and parodontal disease. The necrosis may be limited to the alveolar process, or may involve the body of the bone (fig. 932). Death of a segment of the mandible may result in a solution of its continuity, and so give rise to one form of pathological (' spontaneous ') fracture. (See fig. 935 and p. 924.) How severe a condition can arise will be seen from the following case reported by W. H. Dolamore.[1] It occurred in a boy, aged four, and arose in the region of the mandibular left deciduous molars. A general supporting treatment was carried out with the local use of antiseptic and deodorant mouth-washes. At the end of ten months the necrosed bone was removed, and the extent of the disease can be gleaned from fig. 933. The radiogram seen in fig. 934 shows the extent to which new bone had been formed from the periosteum.

A case is reported by Breward Neale,[2] in which extensive necrosis of the mandible was attributed to a dental operation.

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[1] *Brit. Dent. Journ.*, xxi, 3.

[2] *Trans. Odonto. Soc.*, xxxii, 146.

'The patient, a medical man, consulted a dental practitioner on account of great pain from a mandibular right canine in which the pulp was exposed. At the first visit the pulp was dressed with a devitalising agent; at the second visit a portion of the pulp was extirpated; at the third visit the root and crown were filled, but a flexible drill was broken in the canal. The position of affairs was made clear to the patient, and he went away. On the fourth day he experienced great pain, and he attended and asked for the tooth to be removed, but as it was thought possible to save the tooth an opening was made through the alveolus with a view of relieving the pain. The patient attended the next day, again hoping to have the troublesome tooth removed, but the

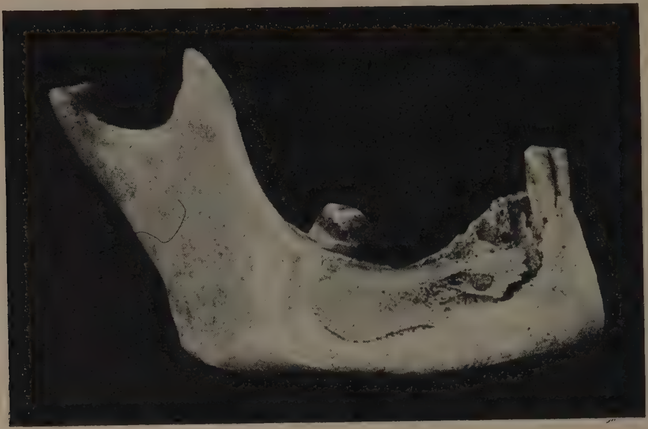

Fig. 932.—Necrosis of the mandible involving the body of the bone. The necrosis was due to infection in connection with the canine.

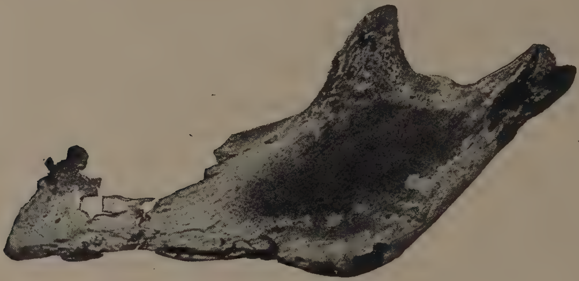

Fig. 933. (From *Brit. Dent. Journ.*)

practitioner still thought removal of the root unnecessary, and excised the crown. On the sixth day, however, the root was extracted, but no relief following, the case passed into the hands of his surgical colleagues.'

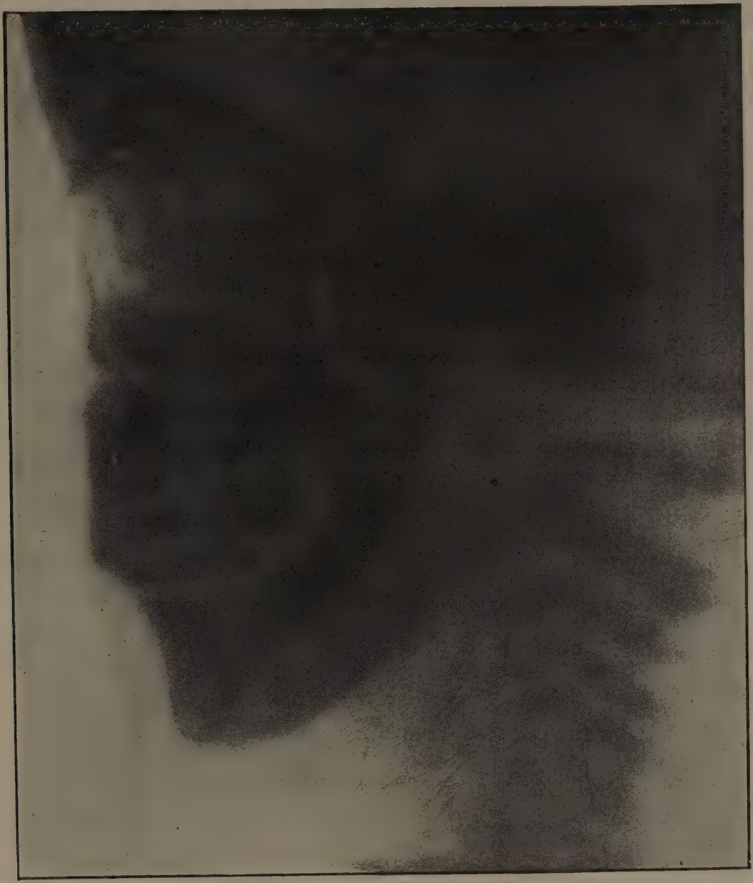

FIG. 934. (From *Brit. Dent. Journ.*)

The patient then went into hospital. The pain and swelling increased and the submaxillary and submental regions became swollen and brawny. Free incisions to the bone gave little relief. The condition became very grave, the whole mandible becoming involved as far as the angle and the outline of the neck being obliterated. The patient fortunately recovered but, at different times, forty-nine sequestra were removed. The average size of the larger sequestra was 1 in. long, $\frac{1}{4}$ in. wide and $\frac{1}{16}$ in. thick.

In children, whose resistance has been lowered by bad hygiene or acute illness, *the infection may reach the bone via the gum.* The necrosis seen in gangrenous stomatitis is an example of this mode of infection.

A third way in which the *bacterial toxins may reach the bone is by the blood-stream.* H. A. T. Fairbank considers that infection through the blood-stream is more common than is generally supposed ; the region of the angle and the ramus are the parts usually affected. The clinical condition is similar to 'acute necrosis' of the long bones. Fairbank quotes the following case as typical of this type of infection : 'A boy was brought to the Children's Hospital, Great Ormond Street, with a brawny swelling over the lower border of his mandible opposite the molar region. Each of the two deciduous molars on that side had a small carious cavity, though there was no sign of periodontitis, and the gums were normal. The swelling was incised through the skin and pus evacuated from beneath the periosteum of the jaw. One of the molars was removed, as it was possibly the cause of the trouble. Except for the superficial caries, the tooth was absolutely normal, and the pulp was healthy.'

The necrosis, occurring as a sequela **of the acute infective fevers,** is probably due to bacterial infection invading the bone from the mouth or the blood, and is in no way a specific effect of the fevers. The septic condition of the mouth present during the acute stages of these fevers, combined with the lowered resistance of the individual, and the liability to injury in the process of cleaning the gums and the throat, all favour the occurrence of necrosis. H. Austen,[1] in an extended experience at the Western Fever Hospital, is of the opinion that necrosis always begins during the acute stage of the illness. In five thousand patients treated at the Western Fever Hospital, all of whom were under observation for at least eight weeks, the cases of necrosis that occurred could all be referred to the acute stage of the illness, not one having arisen during convalescence.

Necrosis is more common in connection with scarlet fever than with measles and is rarely met with in smallpox. In scarlet fever, necrosis is mainly met with in cases in which severe throat symptoms are present, and is usually first clinically recognised in the second and third weeks of illness. Children between the ages of four and six are the most frequent sufferers, but it may occur in younger and older patients.

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[1] *Dental Record,* June 1896.

Austen states that :—

'Scarlatina necrosis of the jaw is most frequent in the lower incisor region on the labial side. It is, however, often seen in the premolar and molar regions on the buccal aspects. In the former situation it is usually symmetrical, less frequently so in the latter. In one case, under the care of a colleague, necrosis occurred on the inside of the ramus, well behind the last molar tooth, and not involving the alveolus in any way. The necrosis may involve the bone forming the sockets of the deciduous teeth only, or more rarely, and especially in the lower incisor region, cause destruction of the bone enclosing the sacs of the permanent teeth.[1] The sequestrum usually takes two or three weeks to separate. The mortality of the cases in which necrosis of the jaw takes place is high, partly from the usually intrinsically severe nature of these cases, partly from aggravation of the symptoms caused by the necrosis. In the worst cases the mouth becomes horribly foul, and the patient soon dies of pyæmia or septic broncho-pneumonia. Two causes may be clinically recognised for this necrosis of the jaw in scarlet fever. Firstly, exposure of the bone by ulceration of the gum, seen in severe stomatitis, already alluded to. Secondly, and more frequently, injury. The latter cause may appear a somewhat remarkable one to anyone not acquainted clinically with this disease.

'For the efficient treatment of the very severe throat condition present in so many cases of scarlet fever, constant applications to the fauces of antiseptics, etc., become necessary, with removal of any secretions likely to decompose or lead to injury. This is done by syringing, spraying, or swabbing, the latter procedure being most effective. These methods, however, necessitate the introduction into the mouth of bone or vulcanite syringe nozzles, spatulas to depress the tongue, occasionally a cork wrapped with lint to gag the mouth open temporarily. However great be the care employed, it is almost impossible in some children to avoid injury to the deciduous teeth. The child will bite the spatula or the nozzle of the feeding vessel, etc., until the teeth become quite loose and fall out, the septic state of the mouth then causing ulceration, and ultimately necrosis of some part of the socket. I have been many times surprised at the very slight pressure with a spatula, or other implement used in examining the throat, that will loosen or extract the teeth in these cases, even when employed with the greatest care and gentleness. Even short of extraction of these teeth I am convinced from clinical observation that necrosis of the alveolus often arises merely from pressure on the crowns, and that not undue in amount. In severe cases in which extensive necrosis appears, I am convinced that it is best to leave the fauces entirely alone, and to feed the child solely with the nasal tube, as if the treatment be persevered in the case will only go from bad to worse.'

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[1] It is possible that some cases where destruction of the permanent tooth sac occurs may be overlooked, owing to the fact that the condition so often proves fatal.

Austen's remarks have been quoted fully because they seem to throw much light upon this hitherto obscure condition and point out that during the acute stage a piece of bone may die from either ulceration or injury. Exanthematous [1] necrosis, as it is called, would therefore seem to be not a secondary specific sequel, but rather a condition which arises during the acute stage.

(*b*) **Chemical Poisons.**—*Arsenious Acid.*—The commonest example of necrosis traceable to chemical poisoning is that which is caused by the escape of arsenious acid used for devitalising the pulp. The poison may act directly on the gum, or may escape through the apical foramen. Usually the necrosis is not extensive, but, with superimposed bacterial infection, a considerable portion of the bone may become involved.

Mercury.—A long course of mercury or the constant inhalation of mercurial fumes may cause necrosis. This form of necrosis is uncommon at the present day, as mercury is not now given in quantities sufficient to produce salivation, and operators employed in the manufacture of looking-glasses are not subjected to the same risks as formerly. Mercury causes necrosis by the conversion of its salts present in the circulation into a sulphide of mercury by sulphuretted hydrogen produced by micro-organisms present in dirty gingival troughs ; the sulphide of mercury, being toxic to the living tissue, sets up an acute gingivitis—leading to periostitis—leading to necrosis (see p. 526 *et seq.*).

At an earlier period mercury used to be given as anti-syphilitic treatment in the following manner : the patient was dosed with increasing doses till salivation occurred, and then kept on two-thirds of that dose for the two subsequent years; at that time oral sepsis and parodontal disease were not understood, and these, in conjunction with the large doses of mercury then given, often gave rise to necrosis of mercurial origin.

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[1] An exanthematous fever is a fever associated with a rash.

Phosphorus.—This form of necrosis was first described by Lorinser of Vienna, while Wilks was the first in this country to draw attention to the subject. It is a severe form of disease and may affect both jaws, though usually only one at a time[1] is affected. In the early stages it is slow in its progress. The condition is one of cario-necrosis and differs in no respect from the same lesion seen in other bones and arising from various causes.

A mandible showing extensive necrosis due to phosphorus poisoning is shown in fig. 935.

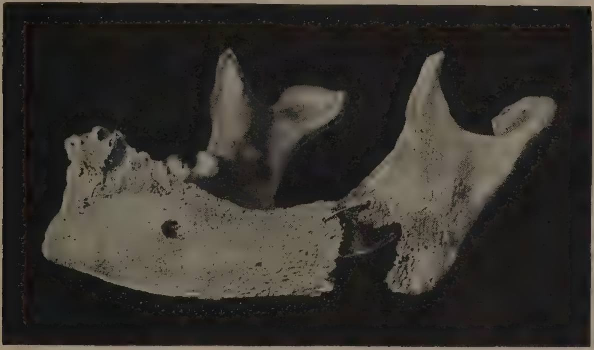

Fig. 935.—Extensive necrosis of the mandible occurring in a lucifer match maker. In this specimen the 'pumice-stone' deposit is not present in the sequestrum.

This form of necrosis is nearly always seen in those subjected to the fumes of phosphorus. It is to be noted that the disease is mainly met with in those engaged in the dangerous parts of the manufacture of matches, namely, 'mixing,' 'dipping,' 'drying,' and 'boxing.'

The fumes emanating from phosphorus consist principally of phosphorus anhydride (P_2O_3) and phosphoric anhydride (P_2O_5). These fumes are probably dissolved in the saliva. (1) The action may be purely local, in which case the irritant finds its way through a septic pulp or wound of the bone and sets up an inflammation of the periosteum, followed by suppuration and necrosis; or (2) the phosphorus fumes produce a general lowering of the resistance of the individual, and also locally, by their irritant action, weaken the nutrition of the bone and render it more liable to attack by micro-organisms.

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[1] In 51 cases collected by von Bibra, 21 occurred in the maxilla, 25 in the mandible, and in 5 cases both jaws were involved.

The sequestrum from cases of this form of necrosis is often peculiar, owing to a deposit known as the 'pumice-stone' deposit. This is found in sequestra from the mandible, but not in those from the maxilla. It is formed from the periosteum, but is so closely adherent to the sequestrum as to be generally brought away with it. A point of interest in regard to the structure of the deposit is that the Haversian canals are larger than in normal bone; they run at right angles to the general direction of the bone, and not parallel to it; they interlace with one another, and in some places form sac-like expansions. Although this peculiar deposit is generally to be found in cases of phosphorus, it may occur in other forms of necrosis.

The *symptoms* of this disease generally commence with toothache, which is at first local and constant, and later becomes more severe and erratic, the pain shooting to the side of the head and towards the shoulder. The disease is at first subacute. The gums become swollen and livid, the swelling and tenderness increase, and suppuration eventually takes place. The skin over the part becomes red, tense, and distended. Bronchial and pulmonary symptoms from irritation may develop, while later, during the advent of suppuration, there is often well-marked pyrexia, accompanied by rigors. The sufferings of the patient are much relieved by the discharge of the pus.

Prognosis.—If the disease is not too far advanced and the treatment is prompt, the prognosis is favourable. In about 83 per cent. of the patients attacked with this disease recovery takes place.

The prophylactic treatment is the most important. The yellow phosphorus is the dangerous form, and its use is now abandoned in this country in match-making. Since the enforcement in lucifer match factories of special rules relating to general hygiene, regular medical and dental inspection, etc., the number of operators attacked by phosphorus necrosis has considerably decreased. In the years 1893 to 1899, previous to the institution of the special rules, thirty-seven cases of phosphorus poisoning occurred among 1,908 persons employed in the dangerous processes, while since the institution of the rules, namely, 1900 to 1907, the incidence has been thirteen in 1,378.

Bismuth.—The soluble salts of bismuth are not used internally, though some of the insoluble ones are, as constituents of bismuth paste (B.I.P.) and constituents of test meals, etc., as they are radio opaque. Occasionally soluble salts of bismuth are present in these as impurities, or an individual may have a bismuth idiosyncrasy ; however it may be, the following case came under notice of one of us : a child aged twelve years had a tubercular knee, which was treated by injection of a teaspoonful of bismuth paste into the joint, as they not infrequently are ; the child became acutely ill, and within a few days the whole of the alveolar bone of both jaws necrosed and was bared of periosteum, several of the teeth dropping out, blue spots occurred on the cheeks and floor of the mouth and necrosis began to occur at those spots when the child died. In cases of bismuth poisoning the breath smells strongly of onions or acetylene.

(*c*) **Trauma.**—Necrosis from traumatic causes is only liable to occur when the injury leads to a comminution of the bone in such a way that the fragment is cut off from its blood supply. Necrosis of small portions of the alveolar process following extraction often occurs in this way. In the majority of cases of necrosis following trauma, however, the death of the tissue is due to septic infection.

(*d*) **Tubercle.**—Tuberculosis of the jaws is a rare and intractable disease. It is usually met with in patients affected with tuberculosis of the lungs. The disease usually starts in other parts of the mouth and spreads to the gums and teeth, more rarely the primary lesion is in the alveolar process. In one case the seat of trouble was a mandibular left second premolar, and the disease, in spite of treatment, involved the whole of the body of the bone on that side. An interesting case is recorded by Partsch,[1] in which the primary lesion was a tuberculous periodontitis, the bacilli having gained access through a carious mandibular first molar.

Injury of the bone from tooth extraction in patients with active phthisis is liable to be followed by necrosis ; the death of the bone may be due to the inability of the tissues to react to the injury, or to infection of the bone by sputum.

According to Carl Zandy,[2] tuberculosis of the alveolar process usually develops between the ages of fifteen and twenty, and affects males more frequently than females.

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[1] *Deutsche med. Woch.*, September 22, 1904 (translated in *Brit. Dent. Journ.*, xxvi, 657).

[2] *Arch. f. klin. Chir.*, iii, 178.

(*e*) **Syphilis.**—Necrosis from syphilis occurs in the tertiary stage, a common site being the hard palate, but any part of the jaws may be affected. The palate may become the site of a gummatous infiltration which, by a process of denudation and ulceration becomes a gummatous ulcer involving the bone, which it ultimately perforates. The mandible is said to be less liable to syphilitic disease than the maxilla.

(*f*) **Radium.**—Radium, its salts or emanation are now frequently used in the treatment of carcinoma of the tongue, lips or any other part of the mouth, and unless the neighbouring bones are protected by metal shields, necrosis may be produced. Sometimes following the application of a single large dose, or of repeated doses, changes in the bone occur, perhaps not apparent at the time, but permanent in their effect. According to H. S. Souttar,[1] 'the resistance of the tissues is greatly reduced, and this may come to light only after a very long period. This is most strikingly seen in the bones, which show no immediate change, but may, years later, fail to respond normally to injury or infection. Thus the removal of a tooth years after irradiation of the tongue may lead to extensive necrosis of the jaw, a matter of great importance in treatment.'

The appearance and clinical signs in this form of necrosis are unusual, bare bone is exposed in the mouth, but it does not appear to be painful or offensive as under other circumstances, nor does the dead portion separate readily as a sequestrum, but may remain as first seen for a prolonged period.

When the surgeon is using radium in the mouth he should work in conjunction with the dental surgeon, in order that adjacent bony tissues may be efficiently protected from the gamma rays by the use of lead shields 1 mm. in thickness. (See Chapter XXXVII.)

(B) SIGNS AND SYMPTOMS.

Necrosis in the early stages simulates periostitis. The gums become much swollen and tender, and suppuration occurs, the pus discharging and leaving sinuses ; the teeth become very loose and pus oozes up the sides. The skin may be shiny, red, and œdematous, and the breath fœtid. In severe cases gangrene may ensue, with fatal results.

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[1] Schorstein Memorial Lecture, 1930.

(C) DIAGNOSIS.

Necrosis is apt to be confounded with epithelioma of the gums which has spread to the maxillary sinus (creeping epithelioma), or it may be mistaken for sarcoma. *Streptothrix* infection also simulates necrosis. Dead bone can be recognised by the grating sensation produced when a probe, on being passed down a sinus, impinges upon the bone. In children the sequestrum frequently involves the permanent teeth, while in cases of phosphorus necrosis it presents a peculiar appearance known as the pumice-stone deposit, which has already been referred to. After removal of the sequestrum in the maxilla, the tissue of repair is usually fibrous in character, no new bone being formed; whereas, in the mandible, a considerable amount of new osseous tissue is formed, and in a notable case recorded by W. Savory (*Roy. Med. Chir. Soc. Trans.*, lvii, 187) the whole jaw was removed from a lad aged eighteen, and six months afterwards, when death occurred, almost a new mandible had formed.

(D) TREATMENT.

In the early inflammatory stages (*i.e.* of general periodontitis, see p. 486) any cause, local or general, should be removed and fomentations applied. A good purge must be given, and, in addition, large doses of iodide of potassium are recommended, opium being added where there is much pain. Should destruction of the bone seem probable, free incisions must be made to relieve tension, while the teeth in the area involved should be removed. The health must be supported, and, if solid food cannot be taken, fluids must be given. *If the bone is necrosed*, but immovable, free drainage should be established and the case left alone for Nature to throw off the sequestrum. If more than one sinus exists, they should be connected and the parts constantly irrigated. As soon as the bone is loose it must be removed with suitable instruments. When the necrosis involves large portions of the jaw, surgical measures may be called for, but should not be adopted until the new bone developed from the periosteum is sufficiently complete to maintain the form of the jaw. For the method of procedure the reader is referred to one of the manuals of surgery.

CHAPTER XXXI

Suppuration of the Maxillary Sinus—Empyema Antri.

The Anatomy of the Maxillary Sinus—the Causes of Suppuration—Bacteriology—Signs and Symptoms—Complications—Diagnosis—Treatment.

The chief disease of the maxillary sinus which comes under the notice of the dental surgeon is suppuration of the lining membrane.

(A) ANATOMY OF THE MAXILLARY SINUS.

The maxillary sinus varies considerably both in size and shape, but these variations can be roughly grouped into three classes, namely, the normal, the small or contracted, and the large or expanded, the size being governed by the amount of tissue between the teeth and the floor of the sinus. The dimensions may be considered as **normal** when the floor of the sinus is approximately level with the floor of the nose; **contracted** when the floor is above, and **expanded** when the floor is below that of the nasal fossa.

The normal sinus extends from the first premolar to the tuberosity of the maxilla. The deepest portion lies opposite the roots of the second premolar and first molar, and from this point forwards and backwards the amount of bone between the teeth and the floor of the sinus increases in thickness. In the expanded sinus the amount of bone which covers the roots of the teeth is decreased, and the roots form projections on the floor of the cavity; the molar roots mostly forming these projections, the premolars less frequently. It is often overlooked that the third molar is closely related to the sinus, but the knowledge of this fact is of the greatest importance in treatment of cases where suppuration of the sinus is suspected to be of dental origin. In the contracted sinus, the amount of tissue between the teeth and the floor of the cavity is considerably increased.

The capacity of the sinus varies greatly. Those of small size may be capable of containing little more than 1 drachm of fluid; while those of the large size may hold as much as 8 drachms. The two sinuses in the same individual may also vary considerably in size. The cavity in many cases is crossed by septa, which, as a rule, run along the floor, the most common situation being between the second and third molars. The septa along the floor may be continued up the outer wall, practically dividing the cavity into two parts.

The sinus may extend into the body of the malar bone. 'Deep pockets or fossæ are met with in various parts of the cavity, particularly at the anterior and posterior corners and along the anterior wall immediately beneath the orbital plate. Sometimes a bony canal terminates in a *cul-de-sac* beneath the orbital plate in the nasal corner of the anterior wall.' The average dimensions of the maxillary sinus are: Vertical height $1\frac{1}{2}$ in., transverse breadth 1 in., antero-posterior depth $1\frac{1}{4}$ in.

Normally, the sinus opens into the middle meatus towards the anterior part of the cavity. In about 10 per cent. of specimens there is an accessory opening or possibly two. These accessory openings, which are usually situated farther back and on a lower level, are, as a rule, larger and more circular than the normal ostium.

A. S. Underwood,[1] who examined about 150 skulls of all ages and both sexes by transillumination, found that the sinus varies considerably in point of size and position, and suggested that its size and position may be entirely governed by the eruption, growth, and subsequent loss of teeth. The molars and second premolars are almost always in relation to the cavity, the first premolar often and the canine rarely.

The relation of the teeth to the sinus depends on the size of the cavity. When the sinus is of exceptional size the canine may project in the floor of the cavity, while, in unusually small cavities, only the second and third molars are in relation with the floor.

Clinically, the size of the sinus may be gauged by the fullness of the facial and nasal walls. As a rule, the more these walls are depressed the smaller is the cavity of the sinus.

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[1] *Brit. Med. Journ.*, August 22, 1908, 463.

(B) CAUSES OF SUPPURATION.

(*a*) **Trauma.**—Injury to the sinus from direct violence, such as a blow on the cheek, or from fracture of the bone, is seldom followed by suppuration unless a bacterial infection is superimposed. Cases of suppuration of the maxillary sinus in infants, reported to be the result of injury at birth,[1] are probably cases of acute osteo-myelitis.

(*b*) **Bacterial Toxins.**—Injury to the mucous membrane by septic infection occurs either from the teeth or the nasal fossa, though the latter is by far the commoner cause of suppuration.

The Teeth.—The roots of the canine, premolars, and molars may project into the sinus, and, when this is the case, direct extension of an alveolar abscess arising from any of these teeth may infect the lining membrane, but usually the second premolar and first molar are the offending teeth. Direct infection may be due to the penetration of a septic tooth or any other foreign body. Such cases are, however, not very common. The antrum is occasionally opened during the extraction of teeth, and this may happen without the knowledge of the patient or the dental surgeon, and though the antrum may become infected in this manner drainage is so good that such openings usually heal up uneventfully.

The accidental forcing of a tooth or root into the antrum during an attempt at extraction may also initiate infection (see fig. 893) and this is perhaps the commonest cause of dental origin. The infection may arise from a dental cyst suppurating and opening into the cavity, but such cases are rare. Another possible source of infection is from parodontal disease; in this condition the septic infection may spread from the parodontal tissues to the cavity. In one case recorded by J. J. Kekwick[2] the tubercle bacillus was present in the pus, the local tuberculosis being started by a periodontitis in connection with a second premolar.

Opinions vary as to the relation which the amount of tissue between the roots of the teeth and the floor of the sinus bears to the liability of infection. Some authors consider that the thinner the bone the greater the liability to infection; others hold that the infection is due more to the infection of the bone generally than to direct extension from the teeth into the cavity.

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[1] See case recorded by D'Arcy Power, *Brit. Med. Journ.*, September 25, 1897.

[2] *Brit. Journ. Dent. Sci.*, May 15, 1895.

The Nasal Fossa.—Extension of infection from the nasal fossa is the commonest cause of suppuration. The acute conditions which occur in the course of influenza and some acute infective fevers are probably due to direct extension of the infection from the nose.

(C) BACTERIOLOGY.

K. Goadby[1] in a series of fourteen cases found various forms of staphylococci and streptococci, viz. :—

Staphylococcus aureus—seven cases.
,, *albus*—eight cases.
,, *citreus*—one case.
,, *viscosus*—two cases.
Streptococcus brevis—eight cases.
,, *longus*—one case.

Of the other organisms identified, the *Bacillus fusiformis aerobius* was isolated twice, Friedländer's bacillus twice, and the *Saccharomyces neoformans* three times.

In acute suppuration of influenzal origin pure cultures of the influenza bacillus may be found. Tilley[2] states that 'As a general rule a mixed infection is present, in which the *Diplococcus pneumoniæ* is prominent, together with the *Staphylococcus pyogenes aureus* and *albus*, *Streptococcus pyogenes* and *Bacillus coli*.'

(D) SIGNS AND SYMPTOMS.

(*a*) **Acute.**—The most typical symptoms of suppuration of the sinus are seen after influenza. There is a feeling of intolerable distension of the zygomatic region, with pain of a throbbing character. The skin covering the bone becomes swollen, red, and very sensitive. Coughing and blowing the nose accentuate the feeling of distension by increasing the tension in the cavity. If the ostium maxillare is patent, there may be a discharge from the nose. The pain is most severe in cases where the opening is blocked, but it is at all times acute, as the inflammatory infiltration of the parts themselves produces pressure on the nerves. When the ostium is blocked an increased tension from pent-up discharge naturally increases the pain. Usually there is nasal obstruction on the affected side and general febrile symptoms are present.

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[1] *Brit. Med. Journ.*, August 22, 1908, 465.

[2] 'The Science and Practice of Dental Surgery,' edited by Norman G. Bennett, 680.

(*b*) **Chronic.**—These cases are often extremely difficult to cure, and may escape recognition for a long time. The most prominent symptom is discharge from the nostril on the affected side. A bad odour is at times noticeable by the patient himself, unless his olfactory powers have been blunted by nasal disease. Pain is not often severe and is not necessarily local. The pain may be referred to the frontal region or to the teeth, or may be felt over the affected sinus. The part may be tender and the patient unable in consequence to sleep on the affected side. There is generally some thickening and capillary injection of the soft parts over the affected sinus. Should the ostium maxillare become blocked when the pus has filled the cavity and is exerting pressure, the symptoms became acute. It must be remembered that pus under pressure in the sinus behaves precisely in the same manner as pus under pressure elsewhere, *i.e.* it finds an exit by the path of least resistance. There is no bulging of the walls or displacement of eyeball or alveolar process. Many cases presenting symptoms which have hitherto been regarded as suppurating sinuses are really cases of suppurating cysts, mostly dental cysts.

(E) COMPLICATIONS.

Suppuration in the maxillary sinus may lead to *necrosis* of its walls, to *infection of the sphenoidal sinuses*, or to *septic meningitis* through venous channels (deep facial and pterygoid plexus).

(F) DIAGNOSIS.

In all cases of chronic suppuration there is some thickening of soft parts over the affected sinus; slight in many cases, but always noticeable. A purulent discharge from the nose, accompanied by a dull, deep-seated pain, is strongly suggestive of suppuration of the sinus. A diagnosis must be made from ozœna; in the latter, the breath is offensive to bystanders but not to the patient, while the reverse is usually the case in suppuration of the maxillary sinus. The presence of septic teeth in the molar or premolar region would assist in the diagnosis. Pain in the frontal region of one side (supra-orbital nerve) should always arouse suspicion of disease of that side; this pain is in some cases severe, even in the chronic types of suppuration.

The *presence of pus may be determined* by several methods :—

(*a*) Cleanse the nasal cavity, and place the patient in a position which brings the ostium lowest. If the ostium is patent, pus will trickle from the nostril. With a nasal speculum pus may be seen in the middle meatus.

(*b*) The *sinus may be punctured* by a trocar and cannula through the canine fossa or nasal inferior meatus, and washed out through the cannula so inserted.

(*c*) *Transillumination.*—Put the patient into a totally dark room and place an electric light in the mouth. Compare the two sides and note :—

(i) Whether the pupils are equally illuminated from within.

(ii) Whether the patient appreciates light equally on both sides (patients can usually answer the question intelligently).

(iii) The amount of light visible around the lower edges of the orbits.

(iv) The amount of light passing through the cheeks.

Conditions influencing Transillumination.

Anatomical.—Transillumination is best seen in patients of spare build, whose palates and nasal fossæ are normal. The degree of transillumination is lessened in cases where the sinus is small; where bony partitions are present; and where the palate is high.

Pathological.—The general transillumination is increased when a cyst is present which has caused thinning and expansion of the bone, provided that the contents of the cyst are clear. Transillumination through the pupil is obstructed in certain morbid conditions of the eyes, while general transillumination is diminished when (1) the sinus contains pus, mucus, etc.; (2) the lining membrane is thickened as the result of a chronic inflammatory process; (3) solid growths are present in the cavity itself or invading it from without.

Transillumination is not an infallible method of diagnosis, but in combination with other methods it is one of great value. Errors are likely to arise in consequence of the varying thickness of the walls of the cavity in the same individual and the presence of suppuration in both sinuses.[1]

Occasionally the signs of suppuration are obscure, as illustrated by the following case [2] :—

A female, aged about fifty-five, very neurotic, complained of severe neuralgic pains of the left side of her face and head, of many years' standing—probably twenty at least. Her peculiar mental condition made it impossible to get a history or to trace any coherence in the distribution or occurrence of her pain. One point only stood out : that she always began her tale of woe by reference to the left maxilla. The mouth was edentulous and all that called for note was a slight fullness of the left maxillary tuberosity, as compared with the right. There was no tenderness observable. Examination of the nose and antral transillumination were negative ; a radiogram showed no sign of teeth. No treatment improved her, and eventually it was decided to explore the left tuberosity, influenced by the slight fullness and persistent reference of pain to that part. On opening up, the crown of a molar tooth was met with, and its extraction was followed by a gush of muco-pus from the antrum. The roots of the tooth were almost absorbed and the remains of the periodontal membrane engorged, showing that the surrounding bone was inflamed.

In this case tangible signs were practically absent, the only indications of trouble being the persistent neuralgia and the tenderness and fullness of the left maxillary tuberosity.

(G) TREATMENT.

(*a*) **Acute.**—The treatment of suppuration in the maxillary sinus consists in giving free vent to the pus and thoroughly draining the cavity, as in the case of suppuration in other parts.

(i) If the cause is septic infection from a tooth, the tooth should be removed ; drainage and lavage should, however, be carried out as far as possible through the nose ; this necessitates an opening being made of sufficient size into the antrum via the inferior meatus. The object of this procedure is to allow the opening into the mouth to close up as soon as it possibly can. If drainage is maintained through the socket of the extracted tooth, which will often have to be enlarged for this purpose and kept open by a vulcanite plug for some time, there is a considerable likelihood of the epithelium from the surface of the gums growing in and joining with that lining the antrum, and once the drainage canal has become epithelialised over it is very problematical if it can be got to heal over again. Even so, owing to the presence of septa dividing the cavity into loculi, opening into the mouth is in some cases a necessity, as the better drainage through the most dependent part of a loculus often gives considerably increased comfort and greatly expedites healing.

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[1] See 'Transillumination of the Antrum of Highmore,' A. Brown Kelly, *Brit. Med. Journ.*, March 25, 1905, 650.

[2] *Royal Dental Hospital Gazette*, i, 49. J. G. Turner.

(ii) If the cause is nasal in origin, the sinus should also be drained through an opening in the inferior meatus.

(*b*) **Chronic.**—The symptoms are often deceptive, more especially when they arise from a nasal cause. Though many cases have been cured by intranasal antrotomy, the operator is in the dark in so far that he cannot see the nature or extent of the disease he is treating. According to Herbert Tilley[1]: ‘These essentials could only be obtained by a full exposure, such as was afforded by the Caldwell-Luc procedure in antral sinusitis, or an external operation, such as Howarth's, for combined frontal sinus and ethmoid suppuration. That complete exenteration of an infected mucous membrane from an accessory nasal sinus might be followed by necrosis of bone or some other complication was a groundless fear, for it was known that within two or three months a new normal ciliated muco-periosteum would replace that which was removed.’

For the technique of these operations the student should consult a work on ‘Diseases of the Nose.’

Vaccine treatment has been used in cases which have not responded to drainage and lavage, but the results have not been satisfactory.

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[1] Semon lecture, *Brit. Med. Journ.*, 1934, ii, 869.

CHAPTER XXXII

Tumours of the Jaws.

Fibrous Epulis—Fibroma—Chondroma—Osteoma—Osteoclastoma—Myxoma—Multiple Neurofibromatosis—Lipoma—Sarcoma—Malignant Melanoma—Multiple Myeloma—Ewing's Tumour—Adenoma—Papilloma—Carcinoma—Endothelioma.

It is proposed in this chapter to give a brief description of the more salient features of tumours of the jaws, with the object of assisting students to diagnose the swellings of the jaw which may come under their notice.

(A) Innocent Connective Tissue Tumours.

(1) Fibrous Epulis.[1]—The fibrous epulis as usually seen is a small mass of connective tissue, of greater or less vascularity according to its age, growing from a pedicle and covered with epithelium of similar structure to that which covers normal gum, and containing inflammatory cells, especially plasma cells; the number of these cells depends largely on the amount of irritation to which the mass has been subjected in the process of mastication and by reason of its environment. They are more common in females than in males, though the reason is not known.

They are inflammatory in origin, and usually the pedicle has origin from between two teeth, most frequently in the mandibular incisor region; calculus is usually present at the necks of these teeth and is the probable source of the irritation which gives rise to them, though the sharp cervical edge of a carious cavity is sometimes so. As chronic irritation always tends to produce fibrosis, so, as the irritation remains, these inflammatory masses become increasingly hyperplasic (*i.e.* they contain an increased

[1] ἐπι = upon, οὖλον = the gum.

number of connective-tissue cells, as opposed to hypertrophic, which means an enlargement of the cells originally present only). They thus become larger.

The pedicle sometimes has origin from the periosteum of bone and sometimes from the periodontal membrane. They may be situated in any part of the dental arch. When they are of long standing the contraction of the connective tissue composing them decreases their vascularity. The epithelium covering them, though similar to that covering normal gum, is laid on very unevenly and may vary from two or three to forty or more cells in thickness; the interpapillary processes on its deep aspect are very irregular and far more extensive than in normal gum and sometimes loop over and join other similar processes. When they have reached such a size that by reason of their position they are in the way of mastication, their surfaces not infrequently become ulcerated. When small they are of a deeper colour than normal gum, owing to their inflammatory vascularity, but when large and un-

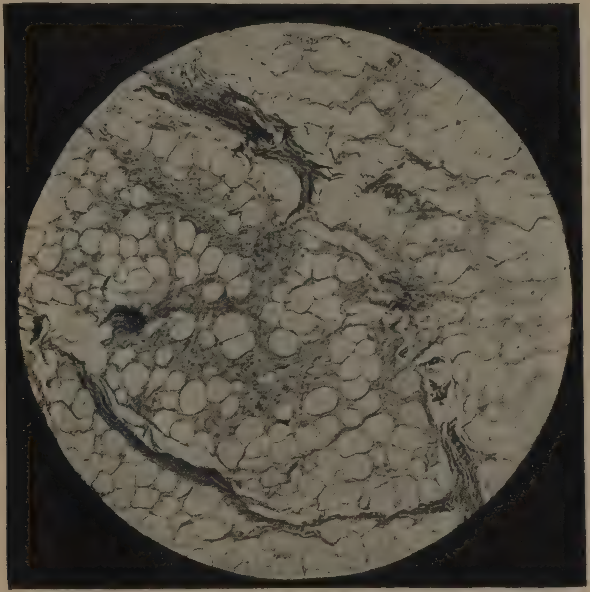

FIG. 936.
(D. L. Radford.) (From *Brit. Dent. Journ.*)

irritated they are often of the same colour as normal gum. When they have reached some size they are, in the absence of inflammatory cells, indistinguishable histologically from fibrous neoplasms (fibromata) of the jaws, but a fibroma may for similar reasons become irritated or ulcerated on its exposed surface and so present inflammatory cells. In addition to the above, spicules of bone are not infrequently present in fibrous epulides; more rarely fat (fig. 936) may be present, and epithelial cells are occasionally present in masses (fig. 937). These latter were believed by Malassez (who first described them) to have their origin from those remnants of the epithelial dental formative organs which may often be seen in sections of normal periodontal membrane and known as the 'rests of Malassez'; they are not connected with the surface epithelium and have been described by J. H. Mummery as being sometimes arranged in the form of cell nests (fig. 938), which may become calcified; occasionally also some of the fibrous connective tissue undergoes degeneration and presents a hyaline appearance (fig. 939).

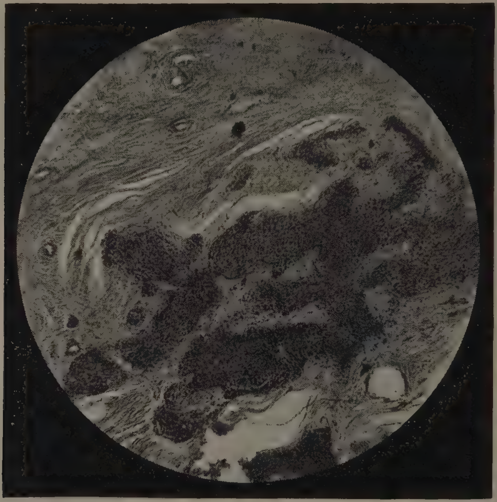

Fig. 937.
(J. H. Mummery.) (From *Brit. Dent. Journ.*)

More rarely, a fibrous epulis may be of the nature of a neoplasm (fibroma); when this is so they do not usually grow from a pedicle, or if they do it is not a slender one. These are perhaps

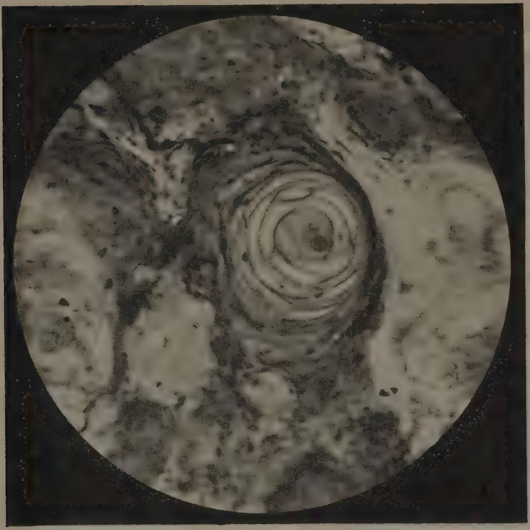

FIG. 938.
(J. H. Mummery.) (From *Brit. Dent. Journ.*)

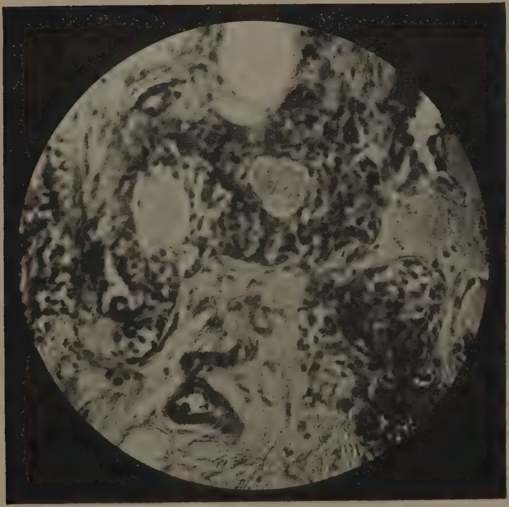

FIG. 939.
(J. H. Mummery.) (From *Brit. Dent. Journ.*)

seen more commonly in children and may be congenital, and occur more frequently in the incisor region.

Treatment.—A fibrous epulis is primarily usually associated with gingival sepsis. If the attachment of the pedicle can be traced to have origin from the periodontal membrane of a particular tooth, removal of that tooth may suffice, as the epulis may come away with the tooth (fig. 940); but if its attachment is uncertain it is better to remove both the teeth with which its pedicle is in relation, when, if its origin is then found to be from the periosteum, that portion of periosteum should be removed together with the growth. When they and their attachment have been completely removed they do not recur. Occasionally they have been known to disappear spontaneously, notably when they have occurred during pregnancy.

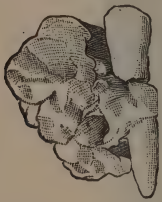

FIG. 940. — Mandibular canine with fibrous epulis growing from the periodontal membrane.

(2) **Fibroma.**—Fibromata of the jaw may be either (*a*) periosteal, or (*b*) endosteal.

(*a*) **Periosteal Fibroma.**—Occasionally swellings composed of dense fibrous connective tissue and covered externally with epithelium are found growing from the jaws; they may have either a sessile or a pedunculated base, and are most commonly found in the mid-line of the mandible and in the premaxilla or at its junction with the maxilla. They sometimes occur quite early in childhood and may even be present at birth.

Fibromata of the jaws are said to occur more frequently in negroid races (among whom Keloid, also a connective-tissue mass, is also common). Nothing is known of the etiology of these tumours and, under circumstances already indicated, they may be histologically indistinguishable from fibrous epulides; indeed it is probable that some tumours described as pedunculated fibromata had their origin as fibrous epulides.

(*b*) **Endosteal Fibroma.**—When this growth occurs in the maxilla it is said to spring from the periosteum of the maxillary sinus. It usually develops slowly and causes a gradual enlargement of the walls by pressure upon the thinner and more readily deformed boundaries—viz. the facial, orbital, and nasal walls (fig. 941). In the mandible the endosteal fibroma causes a gradual expansion of the body of the jaw, and both the external and internal plate walls usually give way. The outline of the swelling is fairly regular.

Considerable interest centres round the origin of the endosteal fibroma of the mandible. J. H. Targett [1] gives an account of a small fibrous growth which was removed from the mandible of a boy aged nine. At the age of seven he had sustained a blow on the chin, and six months afterwards a small swelling, which steadily increased, was noticed in the jaw. On removing the swelling the mandibular nerve was found to pass through the middle of the tumour, the fibrous growth completely surrounding it. Targett is inclined to think that this and other cases of central fibromata of the jaw may originate in the sheath of the mandibular nerve. If this is so they would appear to be of the nature of neuro-fibromata.

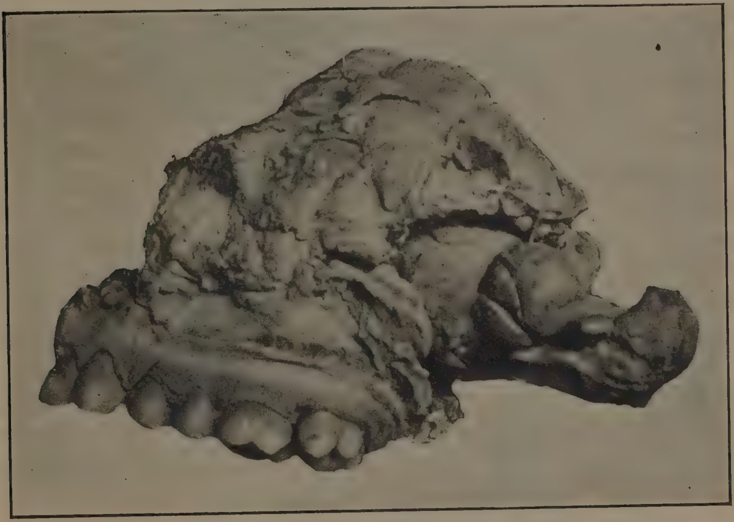

FIG. 941.—Fibroma of the maxilla arising in the maxillary sinus. (Museum of Charing Cross Hospital.)

It is possible that many of the cases recorded as endosteal fibromata belong to the category of fibrous odontomes. As an example we may quote the following case.[2] A female child, aged eight, presented an ovoid, elongated, smooth, hard, and painless swelling on the right side of the mandible. The swelling extended forwards to within 1 inch of the symphysis and backwards beyond the angle of the jaw. The outer plate of the bone was principally bulged, the inner surface being but little altered. The soft parts over the tumour were quite normal, and the teeth behind the canine were missing. On removing the outer plate of bone, the tumour was found to be lying loosely between the plates of the jaw, and was shelled out with little difficulty, being held only at the lower and hinder part, where it lay over a permanent molar tooth. This tooth was removed along with the tumour and was found to be attached to the tumour by a thin membrane. A careful examination showed that this delicate fibrous membrane passed from the outer surface of the tooth root to the delicate connective-tissue capsule of the tumour.

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[1] *Trans. Odonto. Soc.*, xxxiv, 215.

[2] Jordan Lloyd, *Brit. Dent. Journ.*, 1893, xiv, 563.

(3) Chondroma.—Tumours composed of cartilage rarely occur in the jaw. In the maxilla, according to Heath, 'The disease appears as a rule early in life, springing from the surface of the bone or from the antrum, and then making steady progress either externally or internally. The tumour produces absorption of the bone of the maxilla in its progress, and protrudes beneath the skin, which, however, it rarely involves.' Pincus [1] records the occurrence of a small sessile cartilaginous mass in the region behind the maxillary first incisors in a female aged 56; there had been some hæmorrhage, probably aggravated if not initiated by the wearing of a denture.

In the mandible a chondroma may be endosteal or periosteal. The former type causes a gradual distension of the body of the bone somewhat similar to an endosteal fibroma; the latter type may grow to an enormous size, a specimen recorded by Heath weighing 3½ lb. There is reason to believe that some of the cases recorded as chondromata are of a sarcomatous nature (chondro-sarcomata).

(4) Osteoma.—Tumours composed of bone form a most interesting group of jaw tumours. They may be circumscribed or diffuse. They may be composed of cancellous or compact bone.

In the maxilla the whole bone may be affected. An excellent example is shown in figs. 942 and 943. The jaw is expanded generally, but more especially the facial surface. The outer compact plate is smooth except near the infra-orbital foramen, where the cancellous structure forming the tumour appears. Hancock, who removed this growth, refers to the fact that the bone 'yielded to pressure to such an extent as to lead to some doubt as to its osseous nature.'

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[1] *Brit. Dent. Journ.*, 1938, lxv, 168.

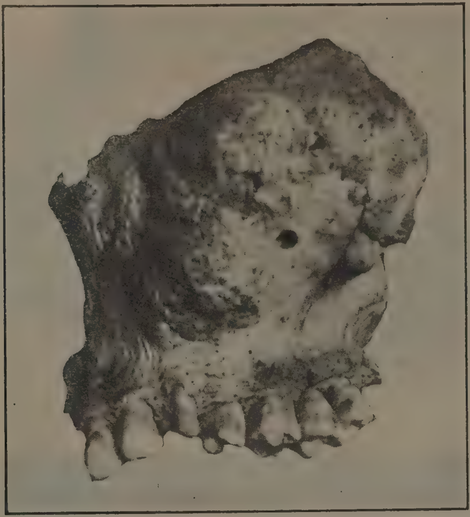

Fig. 942.—Osteoma of the maxilla. (Museum of Charing Cross Hospital.)

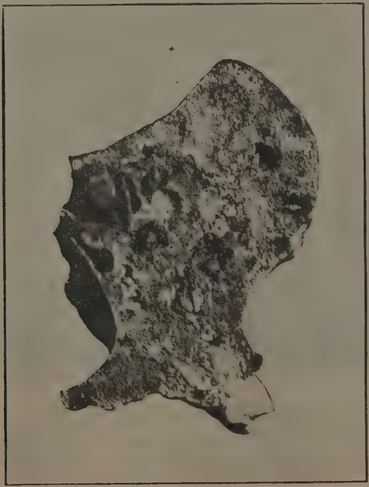

Fig. 943.—Section through tumour shown in fig. 942.

Tumours composed of compact bone and of an ivory consistency have been recorded as occurring in the maxillary sinus. It is possible that many of these are really examples of odontomes.

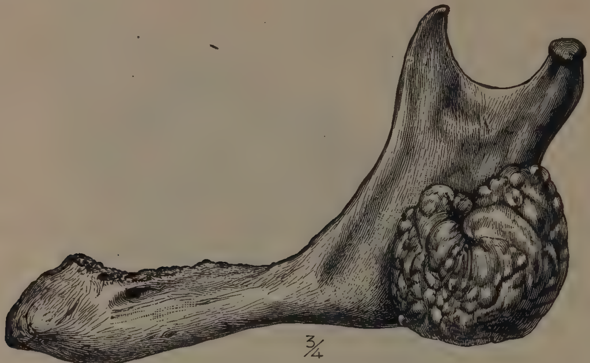

Fig. 944.—A circumscribed osteoma growing from the angle of the mandible. (From 'Tumours, Innocent and Malignant.' J. Bland-Sutton.)

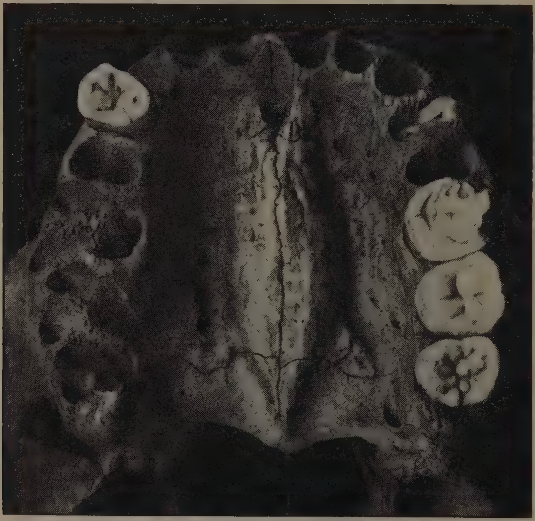

Fig. 945.—Torus palatinus.
Figs. 945 to 947 are from *Proc. Roy. Soc. Med.* (Rickman Godlee.)

Circumscribed osteomata growing from the facial aspect of the maxillæ are often symmetrical.

In the mandible the tumour formed of compact bone is the most common, but occasionally the cancellous variety occurs.

Osseous growths of the mandible are sessile in the majority of cases. The commonest situation is the inner aspect of the alveolar process covering the premolar teeth where they are sometimes bilateral. Other likely situations are the angle of the jaw (fig. 944) and the region of the mental foramen. The whole thickness of the bone may be affected, but such conditions are extremely rare.

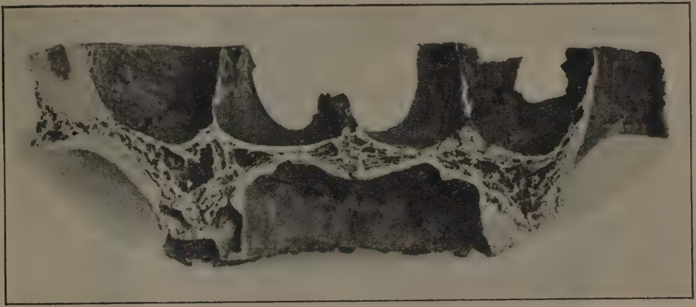

Fig. 946.—Section of fig. 945; the lower table as well as the diploe is thickened.

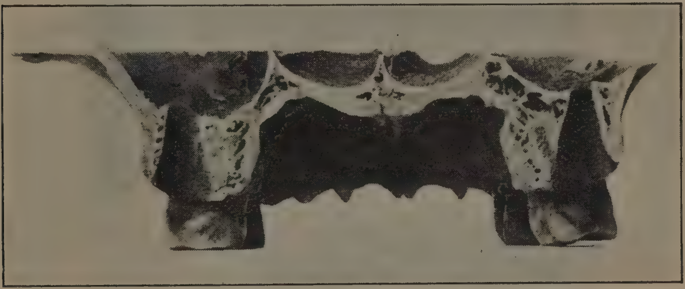

Fig. 947.—Section of torus caused by thickening of the diploe.

Torus Palatinus.—This name is given to a thickening of the bone in the mid-line of the palate, which clinically simulates an osseous growth. It is not a neoplasm. The condition assumes various forms; in some cases the thickening extends almost the entire length of the palate and is symmetrical, as shown in fig. 945; at other times it is asymmetrical. It may be present in children. Sections of bones showing the torus palatinus are given in figs. 946 and 947.

Näcke[1] states that he examined 1,449 individuals, of whom 22 per cent. showed this deformity. He states that it is most commonly met with amongst the insane and criminals.

(5) Osteoclastoma or giant-celled myeloma.—This tumour has also been known as myeloid sarcoma, but objection is laid against its being described as a sarcoma because it is usually benign. It is called myeloma, but this term was in previous use for a round-celled tumour of marrow cells; the constant confusion thus caused may be avoided by distinguishing it as giant-celled myeloma, or better, by calling it osteoclastoma, a name which is gradually gaining popularity. The tumour contains numerous multinucleate giant cells like the osteoclasts which erode bone in normal and pathological conditions.

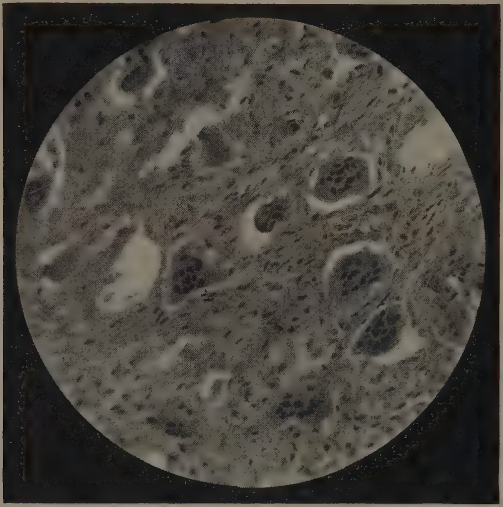

Fig. 948.—Osteoclastoma. Giant cells and small spindle cells. × 120. (J. H. Mummery.) (From *Brit. Dent. Journ.*)

There is probably no bone of the body immune from this growth, but its common sites are the ends of the long bones. It occurs in the jaws, where it may be periosteal or endosteal in origin, whereas in all other bones it is invariably endosteal.

The periosteal osteoclastoma of the jaws is known as *myeloid epulis*. There is no essential difference in microscopic structure between the myeloid epulis and the endosteal osteoclastomata of the jaws and other bones. In the greater part of a tumour, or throughout its whole extent, the tumour tissue is extremely cellular, having very little collagenous (fibrous) tissue. The numer-

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[1] *Neurol. Centralbl.*, June 15, 1893.

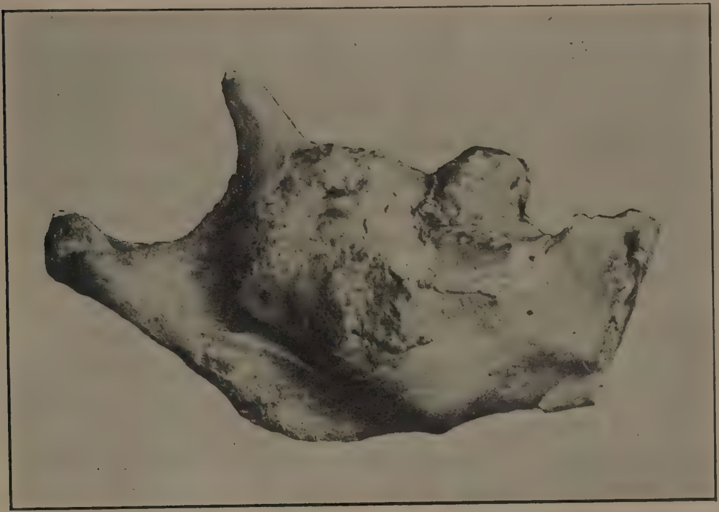

Fig. 949.—Osteoclastoma of the mandible. (Museum of Charing Cross Hospital.)

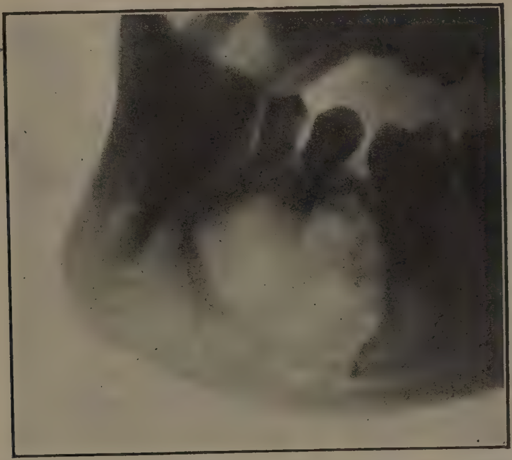

Fig. 950.—Radiogram of osteoclastoma of mandible; the extraction of the permanent first molar six years before had been difficult.

ous giant cells appear to vary greatly in size, though some of the apparently smaller varieties may be sections across the corners of the larger cells. Some of the giant cells are among the largest cells found in the human body. According to G. R. Baxter[1] they vary in size from 20 to 200 μ, and the number of nuclei in any one cell may vary from 2 to 200. The rest of the tumour, between the giant cells, is composed of closely set spindle and polygonal cells like fibroblasts. Hæmorrhage and necrosis, with consequent cyst formation, are common secondary changes ; the hæmorrhage leads to deposit of brown granules of hæmosiderin pigment ; one variety of cyst of bone is a degeneration cyst partly or completely replacing a necrosed osteoclastoma. In parts of some tumours cells are less numerous and fibrous tissue more abundant.

The epulis often has a considerable amount of fibrous tissue as well as very cellular areas with giant cells. It projects from the alveolar margin ; part of its surface, where the epithelium of the gum is still present, is smooth ; in other parts it is granular where the epithelium has been destroyed. The tissue of the epulis varies in consistence, being usually moderately firm. Its cut surface is mottled grey, red and brown, the red and brown colours being due to recent and old hæmorrhage. Necrosis and cyst formation are very rare in the epulis.

In the endosteal osteoclastoma of the jaw there is local replacement of all the bone and marrow of the jaw by soft, grey, red and brown tumour tissue, with production of a characteristic X-ray picture. If the tumour is allowed to grow for a sufficient length of time, an appearance of local expansion of the bone is produced, as the cortex of the bone is eventually destroyed by osteoclastic absorption and a thin shell of bone is formed by osteoblasts in the periosteum, which appears to be bulged outwards and thus forms a fibro-osseous capsule around the growth. At this advanced stage pathological fracture may occur.

Malignant osteoclastoma is very rare and may give rise to metastases in the lungs and other tissues.

Trauma and hæmorrhage have been suggested as factors responsible for initiating the growth of osteoclastoma ; some of those in the jaws have followed tooth extraction. It is alleged, on rather doubtful evidence, that dental sepsis is a causal factor.

According to G. R. Baxter more than half of these growths in the jaws appear between the ages of five and twenty years ; they are rare after fifty years: they occur nearly twice as commonly in females as in males and in 57 per cent. of cases in the lower jaw; in 3 per cent. both jaws are involved.

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[1] *Brit. Dent. Journ.*, 1930, li, 49.

Areas of tissue identical in structure with osteoclastoma occur in the affected bones in some cases of osteitis fibrosa circumscripta and osteitis fibrosa diffusa, but in this last condition the blood calcium is raised. (See also pp. 980 and 982 *et seq.*)

Treatment used to consist of surgical removal, and though this is still occasionally done it is being rapidly superseded, especially as with growths of any size resection of the mandible is disfiguring and attended by other serious disabilities subsequently. It is now known that these and some other primary bone tumours respond readily to radiation therapy, either by means of deep X-rays or radium; under such treatment [1] these growths show gradual reossification, beginning at the periphery and slowly extending centrally; cortical expansions if present may remain indefinitely, but there is no thickening of the cortex.

After curettage of these growths the clot formed becomes organised, and later new bone forms at the periphery and slowly extends inwards, a well-defined bony cortex being formed. The time taken by these changes may extend over several years.

If the swelling of the mandible gives rise to external disfigurement or asymmetry it is well to combine curettage with X-ray treatment, using the former to remove such bone as may account for the asymmetry or deformity.

(6) Myxoma (Myxo-sarcoma).—Some pathologists doubt the existence of pure myxomata, regarding them rather as myxosarcomata of varying malignancy, some indeed being almost benign and not giving rise to metastases. A case of this type was reported by D. W. Daniels.[2] The patient, a woman, aged 27, noticed a hard lump on the side of the mandible near the premolars nine months before coming under observation. Owing to its environment the swelling felt uniformly firm and hard; there was no fluctuation and no obvious swelling in the cheek. The tumour was readily shelled out, leaving a cavity in the bone extending through to the inner plate. The clinical appearances are not easy to differentiate from other affections of the jaw, and radiologically (fig. 951) the appearances may simulate those of multilocular cyst or lipoid granulomatosis (see " Biopsy," p. 989). Under these circumstances the removal of a small portion for biopsy is advisable before definitely deciding on the line of treatment to be followed.

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[1] See A. Brunschwig, *Amer. Journ. of Roent and Rad. Ther.*, 1938, xl, 817, and J. Struthers Fulton, *Proc. Roy. Soc. Med.*, 1939, xxxii, 473.

[2] *Lancet*, 1908, ii, 1747.

(7) Multiple Neurofibromatosis, Neurinoma, von Recklinghausen's Disease, or Molluscum Fibrosum.—This is a rare condition which usually affects the skin of the trunk and limbs, characterised by the presence of innumerable tumours in the distribution of the sensory nerves and related to their skin terminations ; the tumours may be sessile, pedunculated or subcutaneous, are fibrous but may undergo myxomatous degeneration, and contain nerve fibres ; they are unsightly but not painful unless irritated, otherwise the patient may suffer from no ill effects, though some of the tumours may become sarcomatous ; they are generally familial. More commonly **neurofibroma** occurs as a solitary tumour. It is very rare to find these tumours in the mouth ; a case in which this did occur is recorded by G. T. Hankey,[1] who described its clinical appearances as follows : ' There was a soft, fleshy tumour the size of a walnut, and flattened from pressure of the tongue, covering most of the right half of the hard palate. Its colour was the normal pink of the buccal mucous membrane.

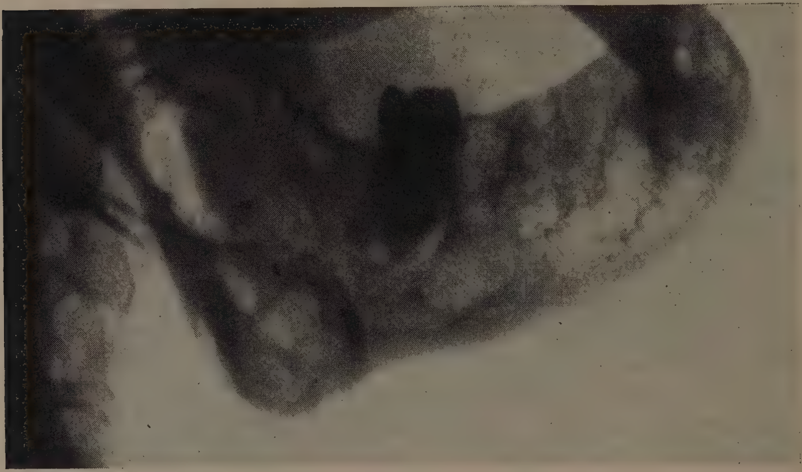

Fig. 951.—Radiogram of left mandible of female aged 46 years. The third molar was unerupted and in intimate association with the growth. Diagnosis by biopsy showed it to be a myxoma (myxo-sarcoma). (G. T. Hankey.)

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[1] *Proc. Roy. Soc. Med.*, 1932–3, xxvi, 959.

It looked slightly œdematous, but there was no sign of inflammation, and it was neither tender nor painful. It was not pedunculated ; the base was almost as large as the lingual surface. There was no enlargement of retropharyngeal or cervical glands.' At operation the tumour was found to be almost gelatinous and extremely vascular. Microscopically it was found to be a fibrous subepithelial nodule with several nerves embedded in the base. This patient had multiple neurofibromata of skin.

(8) Lipoma.—Lipomata of the jaws are very rare, but have been recorded. Clinically they appear as localised lobulated semifluctuant, painless swellings, and give rise to no symptoms save such as may arise on account of their size. They should be removed, and on complete removal do not tend to recur.

(B) Malignant Connective Tissue Tumours.

Sarcoma.

The term **sarcoma** is used to designate a tumour composed mainly of connective tissue cells which in some cases are of an embryonic type. A sarcoma usually presents a homogeneous appearance, the colour varies and depends to some extent upon the vascularity of the growth. Sarcomata vary in their malignancy. Locally, they spread by infiltrating the surrounding tissue and eventually replacing it. A general dissemination throughout the body is brought about by the vascular system.

Sarcomata are classified according to the types of cells of which they are composed ; thus there are round-celled and spindle-celled sarcomata ; a form in which the sarcoma cells vary in size and shape is called a mixed-celled or polymorphic-celled sarcoma, and in some of these there are multinucleate giant cells. The cells are held together by a delicate reticulum, which, with suitable preparation, can usually be demonstrated between the individual cells. Sometimes there are abundant collagen fibres between the cells of a spindle celled sarcoma and then it is called fibrosarcoma, a form which approaches fibroma in structure. The blood vessels in the more malignant sarcomata have very thin walls ; this probably accounts for their great liability to hæmorrhage.

Patients suffering from sarcomata of the jaws are usually young—probably 40 per cent. of them are under twenty years of age. It is possible that chronic irritation or trauma plays a part in their etiology.

The round-celled, spindle-celled, fibro- and mixed-celled sarcomata are met with in about equal numbers. Rare types are the embryo-plastic odontome, the melanotic, the chondro, the myxo, the osteo, and alveolar varieties.

(1) **Round-celled, spindle-celled, mixed-celled, and fibro-sarcomata** may arise either as periosteal or, more rarely, endosteal growths. These tumours may occasionally arise after injury; for example, J. G. Turner[1] records a case where a man received a blow in the front of the mouth, which drove some septic incisors into the bone. Within a very short time he died of a small, round-celled sarcoma, which originated in the injured tissues. These varieties of sarcoma, when springing from the body of the bone, tend rapidly to invade the tissues and give rise to a distension of the bone in all directions, the arch of the teeth being considerably distorted. The periosteal type spreads with equal rapidity in all directions over the surface of the bone and extends down the sockets of the teeth. the bone itself being slowly eroded by the growth.

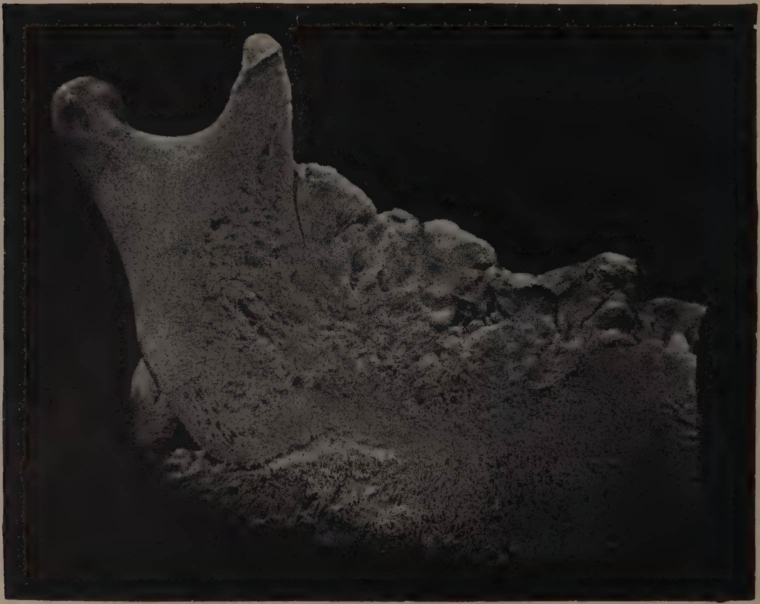

Fig. 952.—A periosteal sarcoma. (J. H. Targett.) (From *Trans. Odonto. Soc.*)

Typical specimens are shown in figs. 952 and 953.

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[1] *Trans. Odonto. Soc.*, xxxix, 112.

(2) Malignant Melanoma (Melanotic Sarcoma).—This type of tumour is rare. In a paper published by New and Hansel [1] twenty-five cases are recorded. The following case [2] illustrates the features of this type of growth. A female, aged 62 years, noticed that an upper denture which she had worn for twenty years became uncomfortable, and on examining her mouth noticed a lump on the gum. When seen there was a lump, with a bluish-black colour simulating gangrene, about the position of a right molar tooth. It projected slightly from the surface; there was no ulceration and no gland enlargement. The growth was removed. The structure was that of a richly cellular melanoma of the spindle-celled sarcomatous type.

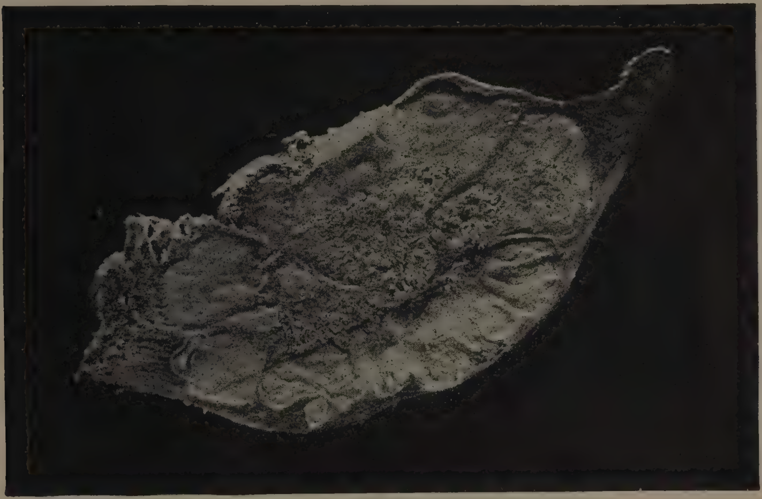

FIG. 953.—An endosteal sarcoma. (J. H. Targett.) (From *Trans. Odonto. Soc.*)

(3) Multiple Myeloma (Kahler's disease) is another rare variety of bone tumour, where tumour nodules tend to occur throughout the skeleton, especially spine, ribs, sternum, pelvis and skull, but may occur in the jaws. It is a tumour of bone marrow, and is composed of closely packed round cells. Many of these tumours were in the past wrongly diagnosed microscopically as round-celled sarcoma of bone. Different varieties occur and are described as being composed of cells resembling the different forms of marrow cell ; the commonest is composed of cells resembling plasma cells and is sometimes known as a plasmocytoma. The disease is commonest in middle age and in females, usually giving rise to painful symptoms at first referred to the spine. The disease is characterised by the presence of Bence Jones protein in the urine and the diagnosis is confirmed by radiological evidence and microscopical examination of tissue.

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[1] *Journ. American Med. Assoc.*, 1921, lvii, 19.

[2] M. J. Stewart and J. Phillips, *Lancet*, 1922, ii, 12.

(4) **Ewing's Tumour (Endothelial Myeloma)** is a very rare and extremely malignant type of tumour which may affect the jaws. It is sometimes called a myelo-sarcoma, and originates from the reticulum cells or endothelial cells of the bone marrow.

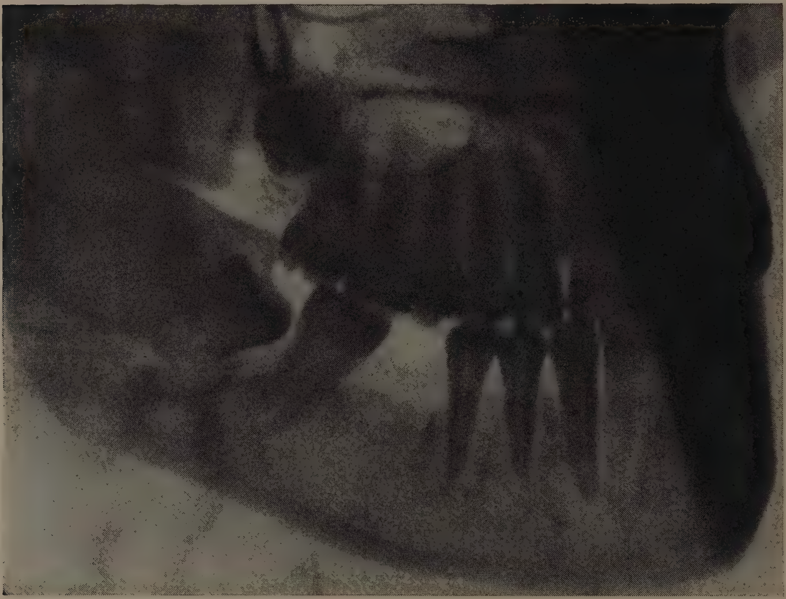

FIG. 954.—Ewing's tumour of the mandible, four months' duration, showing slight diffuse rarefaction of bone in the premolar and molar regions. (G. T. Hankey.)

In a case recorded by G. T. Hankey[1] in a female aged 16, the first sign was tenderness and pain in a mandibular first molar ; this lasted four days and was accompanied by local external swelling of the gums on the fourth day. The swelling was smooth, tense and red ; on extraction of the tooth there was considerable hæmorrhage for twenty-four hours, but no suppuration ; three weeks later granulations appeared growing from the socket and the adjacent teeth became tender, the granular mass gradually advancing along the buccal gum margin anteriorly. When Hankey first saw her eleven weeks after the first symptom there was, in addition, slight puffiness of the face on the affected side, but no tenderness, inflammation or enlarged glands. The swelling was now soft, pink, granular and the colour of normal gum, growing from the molar socket and extending nearly as far forward as the second incisor, where the gum appeared enlarged ; later it involved the sulcus and cheek, causing the cheek and gum to adhere.

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[1] *Proc. Roy. Soc. Med.*, 1933–4, xxvii, 1604.

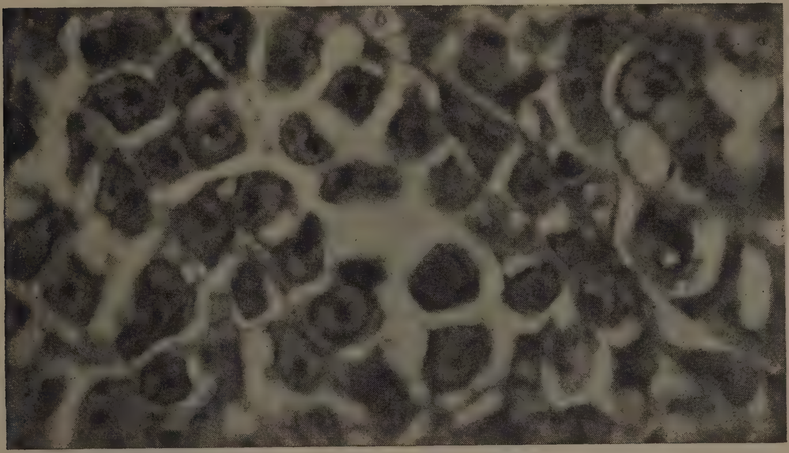

FIG. 955.—Ewing's tumour from gum. × 1200. (G. T. Hankey.)

Eventually it seemed that the growth in the mandible was probably a metastasis from a primary growth occurring on the tibia at the site of trauma from a hockey stick three weeks before the first dental symptom. Despite operative and radium treatment the growth rapidly disseminated throughout the skeleton and caused death within eleven months (figs. 954 and 955). The inciting cause is usually infection or inflammation, but may be trauma.

(C) Innocent Epithelial Tumours.

(1) Adenoma.—This form of tumour is rare in the mouth ; it may arise in the mucous glands of the soft or hard palate, though another view as to the nature of some of these tumours is that they are 'mixed' tumours similar to those in the salivary glands. Adenomata are firm, slow-growing tumours which, on removal, shell out easily, but there are many departures from the typical adenoma, and at times it is almost impossible to distinguish between the adenomata and the carcinomata.

There seems to be a tendency for some adenomata to become malignant. The following case[1] is instructive from the clinical aspect. 'A female, aged 37, noticed a growth about the size of an almond freely movable under the mucous membrane of the hard palate. There was considerable lancinating pain in the tumour, and some carious teeth on that side of the mouth were removed, as they were considered to be the cause of the swelling. There was a discharge of pus from the socket, and the swelling in the palate diminished in size but did not disappear. Three years subsequently the growth was incised, the dimensions of the swelling then being an inch by half an inch. Ulceration followed, and the growth was then freely removed. Microscopic examination showed that it was an adenoma of the palate which had become malignant and infiltrating.'

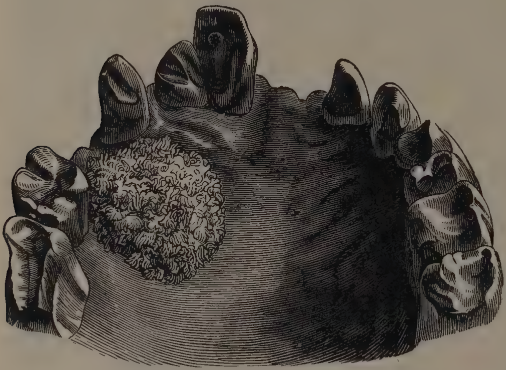

Fig. 956.—Papilloma of the gum.
(From 'Injuries and Diseases of the Jaw.' Christopher Heath.)

(2) **Papilloma.**—Two varieties are met with, the pedunculated and the sessile. The pedunculated are delicate polypoid growths and are usually attached to the soft palate. The sessile papillomata may be found in the muco-periosteum of the alveolar process or hard palate (see fig. 956) and occur occasionally on the mandibular gum.

They vary in size and may be no larger than a millet seed, or the growth may cover the greater part of half the hard palate.[2]

Histologically, they consist of numerous closely set, branching, delicate papillæ, each composed of a central core of vascular fibrous tissue covered with several layers of squamous epithelium similar to that of the mouth, but usually having a superficial stratum corneum; if they are subjected to irritation in the mastication of food, inflammatory cells may also be present.

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[1] *Brit. Dent. Journ.*, xvii, 181.

[2] Arkovy, J., *Trans. Odonto. Soc.*, xiii, 16.

(D) Malignant Epithelial Tumours.

Carcinoma.

The essential feature of a carcinoma is unlimited multiplication of epithelial cells. This cancerous proliferation may start in the epithelial cells covering a surface such as the skin, mouth, stomach or uterus; the growth on the surface at first looks to the naked eye like a papilloma, but usually this superficial papillary part undergoes necrosis from imperfect blood supply and the carcinoma becomes an ulcer, while processes or columns of the proliferating cancer cells extend downwards into the tissue. In other cases the cancerous proliferation starts in the epithelial cells of acini deep in the substance of glands such as the breast or salivary gland, or in epithelial cells such as the liver cells or the cells of the cortex of the suprarenal; then the processes of malignant epithelial cells extend centrifugally from the starting point. As the cancer cells thus invade tissue from a surface, or from the depths of an organ, they destroy the invaded tissue by strangling its elements and destroying its blood supply. But at the same time the irritation of the advancing cancer cells produces a chronic inflammatory type of reaction leading to infiltration with round cells such as lymphocytes, plasma cells and eosinophil leucocytes, and to a proliferation of fibroblasts with formation of collagen fibres and to a proliferation of endothelial cells with formation of new capillaries; this new reactive vascular connective tissue remains in the carcinoma as its stroma, and the cancer cells lie in it in groups. Thus when examined with a microscope a carcinoma is seen to consist of groups of cancer cells separated by vascular connective tissue, but with no connective tissue between the individual cells of a group; the round-celled infiltration is often most abundant around the growing edge of the neoplasm. The amount of connective tissue varies with the rate of growth of the tumour. Thus a slowly growing carcinoma contains much dense fibrous tissue and is therefore hard and is called a scirrhous carcinoma; in a rapidly growing example the connective tissue is minimal, and as such a growth has the soft consistence of marrow or brain it is called medullary or encephaloid.

In carcinomata arising from surfaces covered with squamous or transitional epithelium the groups of cancer cells are solid. This is also the case in some carcinomata arising from columnar-celled glandular mucosa or from the acini or ducts of a glandular tissue; but in other forms of carcinoma arising in the two last-mentioned sites the cancer cells are arranged in hollow tubes or acini resembling those of the tissue of origin and are therefore called adenocarcinoma; the carcinomatous tubes or acini may be very dilated and thus the growth may be cystic.

Carcinomata spread by direct extension into the surrounding tissue, as already indicated. They also spread by the formation of metastases or secondary growths. When cancer invades lymphatic vessels it may grow along them, converting the vessels into very thin solid cords of growth as far as the nearest lymphatic glands, inside which the cancer cells continue to proliferate. More commonly cancer cells are carried in the lymph stream along the vessels, without proliferating in the vessels until they reach the glands, where they commence to form secondary growth; spread along the lymphatic system is known as lymphogenous metastasis; such spread may continue from gland to gland, and the secondary growth may become much larger than the primary. When cancer invades blood vessels the cancer cells may be carried in the blood stream to the lungs, where they may settle down to form secondary growth, and may be carried through the capillaries of the lungs to be disseminated to any tissue of the body; this is called hæmatogenous metastasis. From any of these secondary growths the spread may continue along the lymphatics. Cancer cells are also spread in the central nervous system via the cerebro-spinal fluid, or in the abdomen across the peritoneal cavity, or from one place to another along any other anatomical channel that may be invaded.

(1) Maxilla.

(*a*) **Squamous-celled Carcinoma.**—This neoplasm is commonly known as epithelioma, which is one of the most glaring examples of inconsistency in pathological nomenclature, because epithelioma ought to mean a benign neoplasm of epithelial cells, just as chrondroma is a benign neoplasm of cartilage, myoma a benign neoplasm of muscle, etc. Squamous-celled carcinoma arises from the epithelium adjacent to the maxilla and invades the bone seondarily. A common starting-point is the margin of the gum, generally the outer aspect, adjacent to a tooth which has been the seat of long-continued sepsis. In such a case the chronic sepsis from the tooth seems to have been a determining factor in the growth of the carcinoma. This tumour may originate in the hard palate, and in such cases it can occasionally be traced to the chronic irritation of a badly fitting denture. The disease may also commence in the soft palate, the pillars of the fauces, the tonsils, and the buccal mucous membrane, and thence spread to the jawbone. It is possible that the irritation of a chronic infection, originally syphilitic in character, may cause a malignant growth of the adjacent epithelium.

The squamous-celled carcinoma clinically may at first become evident as an ulcer. There is little doubt that in many of these cases a simple ulcer is present for some time in association with sepsis before malignant change occurs. If, after the removal of all sources of irritation, the ulcer does not rapidly heal, it should be viewed with grave suspicion and immediately submitted to microscopy. In other cases the earliest sign of the disease may be a nodule in the mucous membrane, or a papillary excrescence. But in whatever way it starts, an ulcer with more or less characteristic qualities soon appears. Its edge is hard, raised, and tends to grow luxuriantly over the surrounding tissue, or, as is commonly said, is ‘everted,’ the floor is irregular, and may show irregular warty prominences and deep excavations at the bottom of which the bone may be exposed. The discharge from the ulcer abounds in cancer-cells. A characteristic feature is the extremely fœtid odour emitted from the growth. The ulcer spreads rapidly until a large tract of the jaw may be involved. Cervical glands are early enlarged by malignant deposits, or by chronic sepsis, or by both.

A curious form of squamous-celled carcinoma occurs in the maxilla, to which the name ‘boring epithelioma’ has been given. As the name implies, this type of cancer tends to burrow into the substance of the jaw-bone or even into the sinus, showing from the surface very little, if any, evidence of its grave nature. The onset of this disease is insidious, and the symptoms very indefinite. It often appears to originate in chronic sepsis, as the following case shows: A patient,[1] a female aged 35, had had suppuration in connection with a maxillary left second incisor for a period of two years. The alveolar process retained its natural contour; there was no swelling or redness, but about half to three-quarters of an inch above the neck of the tooth there was a sinus, the edge of which was not swollen but was covered with sodden epithelium.

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[1] *Trans. Odonto. Soc.*, xxxiii, 231.

The tooth was loose and a little tender, and, on pressing the parts, a small quantity of thickish pus was discharged through the sinus. The tooth was removed with a little difficulty, and adherent to it were ragged masses of tissue. On examination these proved to be carcinomatous in character (fig. 957). At the operation the growth extended from the right first incisor to the left first molar, and had only invaded the alveolar portion of the bone.

(*b*) **Carcinoma of Maxillary Antrum.**—It might be expected that carcinoma arising from the cells lining the maxillary antrum would be a columnar-celled carcinoma (adenocarcinoma), but although this is sometimes the case it is more commonly a squamous-celled carcinoma, which here, as less commonly in the gall bladder and the bronchi, arises from columnar epithelium by a process known as metaplasia. The carcinoma tends to fill the cavity of the antrum completely and finally erode one or more of its walls and invade the adjacent soft tissues. When confined to the cavity, the symptoms are very indefinite and are generally those of a persistent and intractable neuralgia. At an early

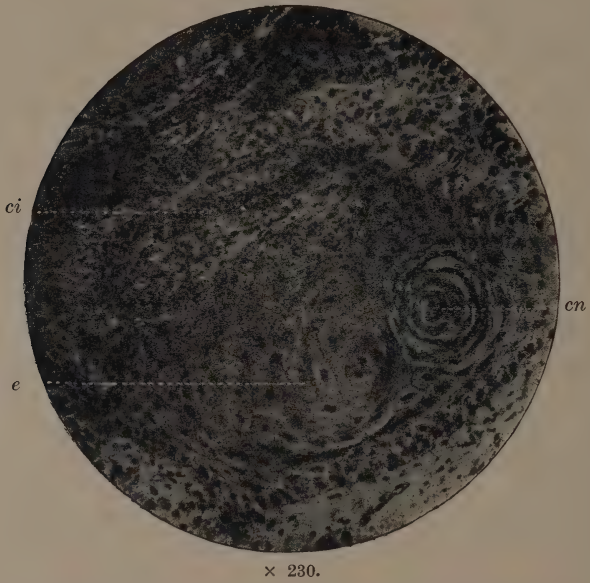

FIG. 957.—*cn*, cell-nest ; *ci*, small-celled infiltration of the tissues ; *e*, islands of epithelial cells. (From 'Histology and Patho-histology of the Teeth.' A. Hopewell-Smith.)

period, if the maxillary sinus were transilluminated from the mouth, opacity might suggest the cause of the neuralgia. Generally speaking, the symptoms are in abeyance until the cavity is filled with the growth and pressure is exerted upon one of its walls. The growth may merely press upon the walls of the cavity and cause distension, or may erode through the boundaries of the cavity and infiltrate the soft tissues. For instance, the orbital plate may be pushed up, and perhaps some limitation of the movements of the globe of the eye may be seen; the facial surface may be encroached upon and a prominence of the cheek occur, with or without infiltration of the soft parts; or, should the posterior wall of the cavity be affected, a swelling may appear in the temporal region of the head above the malar arch. Inspection through the anterior nares may show a bulging of the inner wall of the maxillary sinus, or even a fungation of the growth into the nasal cavity. The teeth on the affected side may loosen and fall out, the sockets becoming filled with the growth. The cervical glands will sooner or later become enlarged.

Eve maintained that many of the growths of this type have their origin from 'rests' of the germinal epithelium of the teeth, and this might equally apply to the other types of carcinomatous growth which occur in the maxilla.

(*c*) **Spheroidal-celled Carcinoma.**—This arises at the back of the tongue and in the pharynx. It is also known as lympho-epithelioma because it is supposed to have a special tendency to take origin from squamous epithelium under which there is lymphoid tissue. But it appears merely to be a type of cancer which arises occasionally from any sort of epithelium in any part of the body, a type which is called undifferentiated, dedifferentiated or anaplastic carcinoma, because the cancer cells do not resemble (do not show differentiation towards) the epithelial cells from which they arise, but are rounded cells; and it is thought that these rounded cells may represent a throw-back to the type of cell found in the early embryo, a theoretical phenomenon to which the term anaplasia has been given. Such growths are very malignant and are usually exceptionally radio-sensitive.

(2) Mandible.

Squamous-celled Carcinoma.—As in the maxilla, the growth arises from the adjacent epithelium, and, secondarily, invades the bone. It may originate at the gum margin, when chronic sepsis appears to play an important part in its causation; or it may commence in the floor of the mouth, the tongue, or the buccal mucous membrane, or the jaw may be secondarily invaded from the lower lip. At times the site of the growth may be determined by irritation from an ill-fitting denture and at others by the impingement of the heat and smoke from smoking a short pipe.

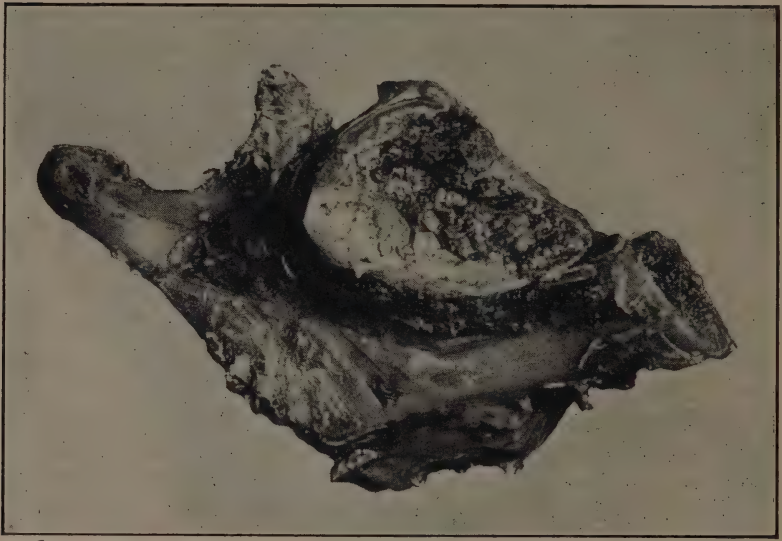

Fig. 958.—Squamous-celled carcinoma of the mandible. (Museum of Charing Cross Hospital.)

A squamous-celled carcinoma arising in the anterior extremity of the sublingual sulcus (sublingual carcinoma) is of importance to the dental surgeon, as the ulceration may be mistaken for a simple ulcer arising from the irritation of a denture. The disease starts as a small nodule, or warty ulceration, in the floor of the mouth; ulceration follows and the disease spreads rapidly between the root of the tongue and the concave surface of the arch of the mandible. The surrounding tissues are speedily infiltrated ; the tongue becomes fixed and the salivary ducts obstructed by pressure, or laid open by ulceration.

The early recognition and treatment of carcinoma are most important, and, as the condition may come under the notice of dental practitioners in its early stages, its clinical signs should be thoroughly understood by them. All patients over thirty with ulceration of recent origin should receive close scrutiny. The following is a typical history. A. B., aged 55, stated that two teeth in the maxillary left molar region became loose and fell out, and that he had since been troubled with a nasty taste in the mouth. An examination of the teeth sockets showed them to be filled with a carcinomatous mass. On another occasion, a man, aged 47, complained of pain in the maxillary left molar region. He had received a blow in this region eighteen years previously, and the first and second molars had recently loosened and fallen out. The wound was filled with a mass of unhealthy-looking tissue ; necrosed bone could be detected, and there was a profuse fœtid discharge. The patient had acquired syphilis twenty-five years previously. In this case, although the history was suggestive of a gummatous infiltration of the bone, the possibility of carcinoma could not be neglected. Microscopical examination showed merely granulation tissue. Under anti-syphilitic treatment and local irrigation the condition cleared up.

(E) Endothelioma.

Endothelial tumours arise in connection with blood- and lymph-vessels. The benign variety is known as angeioma. Hæmangeioma is composed of blood-containing spaces and is one of the commonest tumours of the human body, while lymphangeioma is composed of lymph-containing spaces and is comparatively rare. Hæmangeioma is sometimes a congenital tumour, being present at birth, and then may exhibit no neoplastic tendency, only increasing in size at the same rate as the normal tissue. The birth mark in the skin that is known as a port-wine stain is an hæmangeioma. Vascular nævus is another name for hæmangeioma ; a cavernous hæmangeioma is composed of wide blood spaces and a capillary hæmangeioma of minute blood spaces. The blood-containing spaces that constitute the growths are usually not vessels as their walls contain only fibrous tissue ; there is a rare type composed of vessels with muscle and elastic fibres in their walls. The microscopical appearances of an hæmangeioma are shown in fig. 959. Hæmangeiomata occasionally occur upon the gums. They usually present a smooth surface, and vary in colour according to the degree of oxygenation of the blood in them ; on pressure being applied they become pale, rapidly returning to their original condition on removal of pressure. They are more common in the incisor region, though they may occur in the region of the molars. Starting as a small red spot, they gradually spread between the teeth and extend principally along the margin of the gum, both in front and behind the teeth. These growths bleed freely when touched with the tooth-brush, and in one case under notice the hæmorrhage was severe. In the case of cavernous hæmangeiomata, the tumour may attain to a large size and involve the mucous membrane of the gum, cheek, and lip.

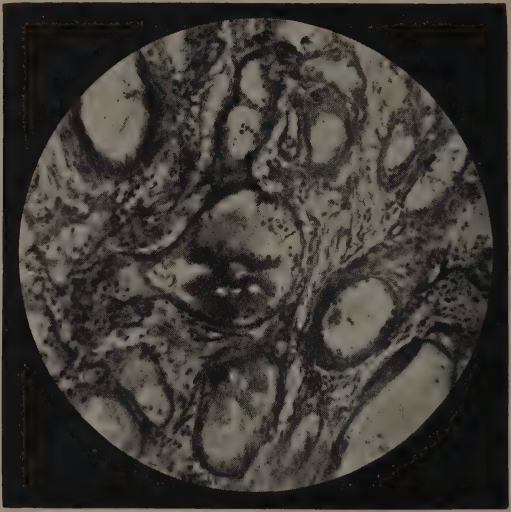

Fig. 959.—Hæmangeioma. From a growth on the gum. × 120. (J. H. Mummery.) (From *Brit. Dent. Journ.*)

In a case (fig. 960) recorded by H. Chapman,[1] an hæmangeioma covered the area corresponding to the distribution of the second division of the fifth nerve. 'From the right incisor to the remains of the first molar the tumour presented a smooth, venous-coloured surface; posteriorly to the latter tooth the colour was lighter, and the surface resembled a cauliflower in appearance. In this region two or three lobulated masses of nævoid tissue were present.'

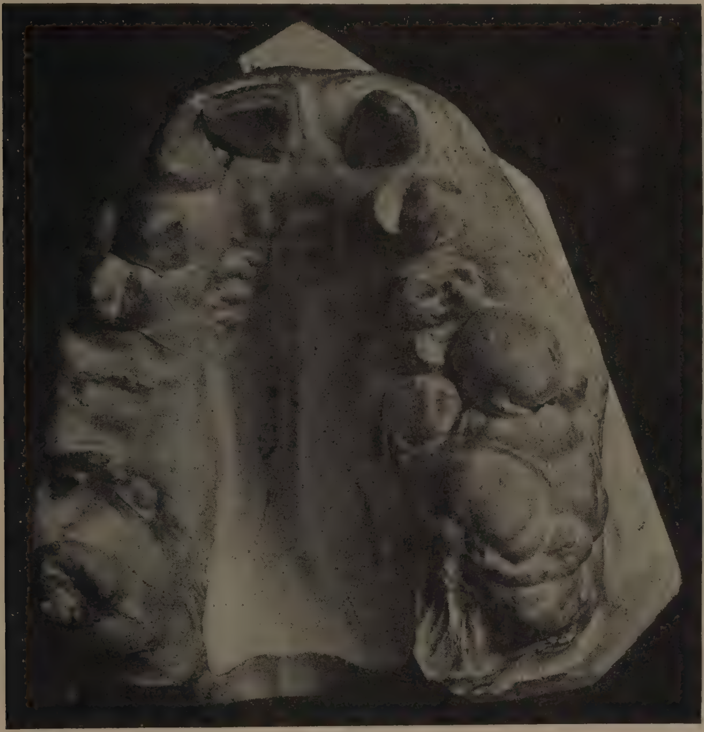

FIG. 960.
(H. Chapman.) (From *Brit. Dent. Journ.*)

Hæmangeiomata are rare on the palate. A case is reported by Treves[2] of a small hæmangeioma about the size of a cherry situated between the maxillary right incisor teeth; it was attached to the periodontal membrane and came away with the second incisor.

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[1] *Brit. Dent. Journ.*, 1915, xxxvi, 472.
[2] *Brit. Dent. Journ.*, ix, 252.

In a case recorded by H. A. T. Fairbank[1] there was a bright red vascular growth behind and slightly round the sides of a maxillary left second premolar. There was no enlargement of the alveolar process and the glands were not affected.

Lymphangeiomata of the gums or jaws are rare.

Malignant endothelioma is very rare and is difficult to distinguish both clinically and microscopically from sarcoma. It is composed of closely packed oval-nucleated endothelial cells like the cells of spindle-celled sarcoma, though in parts it may differentiate to the extent of forming blood spaces and thus give the clue to its endothelial nature.

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[1] *Proc. Roy. Soc. Med.*, iv (Odonto. Sec.), 12.

CHAPTER XXXIII

Diagnosis of Swellings on or in the Jaws.

Swellings on or in the jaws are not always easy to diagnose, and it is often impossible to determine their exact nature without making a preliminary incision, puncture, or microscopical examination.

SWELLINGS OF THE MAXILLA OR MANDIBLE MAY BE FLUID OR SOLID.

(A) The differential diagnosis of the fluid swellings lies between: abscesses, cysts of eruption, dental and dentigerous cysts, multilocular cysts, blood cysts in myelomata, mucous cysts, gummata and naso-palatine cysts.

Acute abscesses.—With these, whether dental or parodontal, the short history and acute signs and symptoms are very typical; parodontal abscesses, whether acute or chronic, may arise in connection with live teeth.

Chronic abscesses, if dental in origin, show the presence of a dead tooth or root, and whether dental or parodontal have a more or less prolonged history; they increase or decrease in size from time to time, the signs of inflammation may not be very marked, in radiograms they do not as a rule show such sharply defined outlines as do cysts.

Cysts of eruption are always superficial and lie over a tooth which should be about to erupt; **they occupy a portion of the alveolar border,** and if they are large and contain much fluid they appear bluish. They give rise to no symptoms.

Dental cysts.—Usually there is a pulpless tooth or root present, which may, however, have been extracted; there are no signs of inflammation, there is a history of a slow and continuous increase in size, they may occur up to any age and usually show a sharply defined outline in radiograms. If a cyst should become infected and discharge, it becomes to all intents and purposes a chronic abscess.

Dentigerous cysts are not as a rule found after the twentieth year except in the case of third molars; often there is a pulpless deciduous tooth present. A tooth is always absent from the erupted series unless the tooth involved in the cyst is one in excess of the normal number.

A radiogram is diagnostic; such cysts may become infected and discharge, when the appearance may be that of a tooth erupting into or contained in a chronic abscess. Unless infected there are no signs of inflammation and there is a slow continuous increase in size.

Multilocular cysts.—There are no signs of inflammation, the swelling is of very slow growth, and often presents a nodular or lobulated surface, usually first noticed at from eighteen to thirty-five years of age; they have been said to occur more frequently in females, but the sex difference, if any, is probably but small. There is frequently no tooth absent from the normal series. In radiograms separate cysts may be seen separated by fibrous or osseous septa; by far their most frequent site is about the mandibular third molar region and they often extend up the ascending ramus and into the coronoid process along the less dense portion of the bone. Fluctuation cannot usually be elicited.

Blood cysts in myelomata may occur in growths seen on the alveolar ridge or any part of the jaws; they are distinguished by the maroon colour of their immediate environment and its usually nodular surface; the growth, apart from the possibility of a blood cyst in it, may cause a yielding of the outer alveolar plate.

Mucous cysts.—These lie very superficially and do not involve any bone deformity, except that that type of mucous cyst which occurs in the antrum may cause a bulging of the outer alveolar plate. They are all painless, smooth, and globular. The mucous cyst of the antrum may, as the bony wall thins, give rise to egg-shell crepitations. They may not be fluctuant.

Gummata.—Gummata may occur on any aspect of the jaws or palate as swellings which fluctuate before they ultimately break down to form gummatous ulcers. As they are syphilitic lesions, other signs of syphilis should be looked for and the history inquired into. They disappear rapidly under antisyphilitic treatment. The Wasserman reaction should be tested.

In the case of all fluid swellings of both maxilla and mandible the outer plate of bone yields before that of any other aspect, although ultimately other aspects may also yield ; but in the cases of the mandibular third molar, the maxillary second incisor, and the palatine roots of notably the maxillary second premolar and maxillary first permanent molar, swellings having their origins from these often present on the lingual or palatal aspect of the bone as being the direction of least resistance. Fluctuation may be elicited in all fluid swellings if they are near enough to the surface ; if the bone over them is much thinned a typical crepitant crackling may be noticed. The alveolar border is not involved except in the case of cysts of eruption and (possibly) blood cysts in myelomata, and gummata just before they are about to ulcerate.

Naso-palatine cysts.—These occur in the premaxillary region, originating either in the region of the anterior palatine fossa or in the lines of union of adjacent embryonic processes ; they are mostly dermoids though some may be retention cysts. They are not very common and are often discovered by chance on routine radiological examination. If small, they give rise to no signs or symptoms, and doubtless many have been mistaken for dental cysts in the past, but no causative pulpless tooth may be present and all teeth may have live pulps. They usually contain sebaceous material, sometimes of a stiffish consistency. They may become irritated and stimulated to further growth by the wearing of a denture ; sometimes they become infected and then simulate abscesses. They are not of dental origin and may be first noticed as swellings on the floor of the nose or occasionally in the labial sulcus.

(B) The differential diagnosis of the solid swellings lies between :—

(1) Innocent swellings.—Fibrous epulis, Fibroma (periosteal or endosteal), Giant-celled Myeloma, Osteoma (compact or cancellous). Solid odontomes (composite or connective tissue), Papilloma, Adenoma, Lipoma, Chondroma, General Hyperplasia of Gum, Osteitis fibrosa diffusa (or cystica), Osteitis fibrosa circumscripta, Leontiasis ossea, Lipoid granulomatosis, Actinomycosis, unerupted misplaced teeth.

(2) Malignant swellings.—Carcinoma, Sarcoma.

(1) Innocent swellings. Fibrous epulis.—This grows from a pedicle attached between two teeth ; usually there is gingivitis and some calculus about these teeth ; they usually grow slowly and unless irritated are coloured like normal gum, except when very small; they are commoner in women and in the mandibular incisor region.

Fibroma (Periosteal).—This may be sessile or pedunculated; in the latter case it may be indistinguishable clinically and histologically from fibrous epulis.

(Endosteal.)—This when occurring in the maxilla may have origin from the periosteum of the antrum. In the mandible they cause a gradual increase in thickness of the bone, they may occur at any age, they are difficult to differentiate clinically from sarcomata, but their rate of growth is slower and there are no metastases in neighbouring glands or elsewhere. They are sometimes (? usually) of the nature of neuro-fibromata.

Osteoclastoma (Giant-celled Myeloma).—The distinguishing features are its maroon colour and lobulated surface; it may cause a bulging of the outer alveolar plate, and owing to internal hæmorrhage give rise to fluctuating blood cysts; it often causes teeth adjacent to it to become loose, and on their loss shows itself growing up into a tooth socket. It does not give rise to metastases. Radiograms show that bone has been eroded and given place to the neoplasm. The blood calcium should be estimated as a matter of routine in all cases where swellings present myeloplaxes; it will be found to be normal in the case of osteoclastomata but raised in cases of osteitis fibrosa diffusa; the lesions of these two diseases are often identical histologically, and the estimation of the blood calcium is the only constant factor in their differential diagnosis (see also p. 958 *et seq.*, and osteitis fibrosa, on p. 982 *et seq.*).

Osteomata.—These occur more commonly in the mandible and have their favourite sites: they occur most commonly on the inner aspect of the alveolar plate adjacent to the mandibular premolars where they are sometimes bilateral at the angle of the mandible and in the region of the mental foramen. They have also been recorded as occurring in the maxillary sinus. They are hard, usually sessile, and give rise to no signs or symptoms except such as may arise on account of their size. In radiograms increased density is notable where they occur. Their growth is very slow.

Usually they occur as local projections from the bone (exostosis), and consist of a dense outer shell with bone of a cancellous character internally; sections of this bone are not easily differentiated from osteitis fibrosa, but should show fatty or hæmatogenous marrow between the bony trabeculæ. Very dense (compact or ivory) osteomata are said only to occur on the skull.

Solid Odontomes.—These give rise to hard swellings of the jaws, frequently a tooth is absent from the normal series, in the course of time many composite odontomes tend to erupt; when this occurs the presence of an abnormal mass or masses composed of calcified dental tissues makes their diagnosis easy. If unerupted their appearances in radiograms usually make their diagnosis easy, the appearance of a calcified mass or masses unlike any normal tooth being pathognomonic of some varieties. They are painless and of slow increase till in their endeavour to erupt a sinus is produced over them which becomes infected and so gives rise to painful suppurative symptoms.

Connective Tissue Odontomes.—In the case of **fibrous odontomes** a tooth may appear to be absent from the normal series, its eruption being prevented by overgrowth of the fibrous tissue of the tooth sac. They are very rare in man but commoner in animals, and are believed to be a result of rickets. They may be multiple and symmetrical. Radiograms and an enquiry as to the history of the individual are therefore of importance.

Cementomata, in so far as their clinical history is known, give rise to no signs or symptoms other than the gradual increase in size of a hard swelling till secondary symptoms occur; these are mostly due to suppuration following infection. They may reach a large size. Radiograms should prove invaluable as an aid to diagnosis.

Papilloma.—This, as a sessile or pedunculated growth, occurs on hard palate, the hyperplasic papillæ give it a warty appearance which is diagnostic. Another variety which occurs on the soft palate is a pedunculated polypoid growth. The only signs or symptoms they give rise to are by reason of their size or their giving rise to irritation when dentures are worn or owing to the movements of the tongue on food during mastication.

Adenomata occur mostly on the hard or soft palate; they may also arise from mucous glands in gum tissue. They are not common and sometimes are mixed tumours rather than pure adenomata. They are painless swellings, and on the hard or soft palate are often clinically difficult to differentiate from carcinomata, although they are as a rule of slower growth. They do not give rise to metastases and have been confused with "adamantinomata."

Lipoma on the jaws is very rare. It occurs as a painless, rather superficial, semifluctuant, lobulated swelling of slow growth, with no other signs or symptoms save as are due to its size.

Chondroma of the jaws is very rare. In the mandible they may be endosteal or periosteal, the former causing a gradual distension of the body of the jaw similar to that seen with endosteal fibroma; the latter, according to Heath, may grow to a large size. In the maxilla, according to Heath, they appear early in life growing from the surface of the bone or from the antrum.

General Hyperplasia of Gum.—This is a general increase in the amount of gum tissue, occurring in both maxilla and mandible simultaneously, though sometimes involving one side more than the other; it sometimes is noticed in early childhood, occasionally in several members of the same family. Teeth are sometimes unable to erupt through the hyperplasic mass and remain permanently buried. Sometimes undue hairiness is an accompanying factor. It is thought that hyperpituitarism may govern its incidence.

Osteitis Fibrosa Diffusa (or **cystica**), also called **von Recklinghausen's disease of bone, Fibro-cystic disease of bone** and **Generalised osteitis fibrosa.**—The first sign of this disease may appear in the mouth as a uniform localised painless swelling, chiefly in the maxilla.

This disease is caused by hyperparathyroidism, and characterised by multiple areas of absorption of bone throughout the skeleton, these areas being replaced by fibrous tissue in which new bone may be formed; cysts may develop in the fibrous tissue. In some areas osteoclastomata occur instead of fibrous areas, and these may be identical histologically with the neoplastic giant-celled myeloma, and as the first swelling to be noticed may occur in the jaws, and if so, usually in the maxilla, it is important to differentiate between them.

Apart from radiological examination of the bony skeleton, the chief point in clinical differential diagnosis rests on the estimation of the blood calcium. The blood calcium should normally be from 9·5 to 10·5 milligrammes per 100 c.c., and in this condition is raised perhaps to 12 or 14 milligrammes. As this is the only constant factor in the differential diagnosis of these lesions from those of osteoclastomata and osteitis fibrosa circumscripta, it should be tested in all cases of growths presenting myeloplaxes as a matter of routine. Clinically, the signs and symptoms may

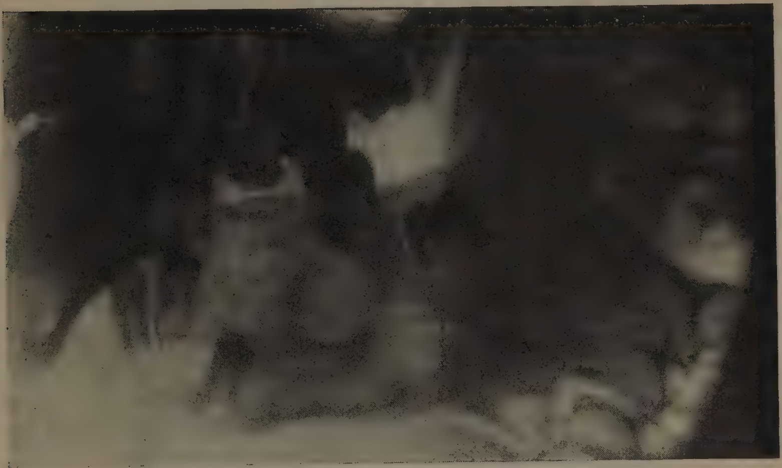

FIG. 961.—Osteitis fibrosa circumscripta in the mandible of a male aged 8 years. (G. T. Hankey.)

FIG. 962.—Osteitis fibrosa circumscripta, section from fig. 961. (G. T. Hankey.)

be very similar to those of cancellous osteoma. (See also pp. 958 *et seq.* and 980.)

One has noticed that in the majority of cases of hyperparathyroidism one has seen and in which calcium salts had disappeared freely from the bones so that they had become more or less plastic, the teeth have appeared more translucent than was normal to the age of the individual, though caries did not appear to be increased.

Osteitis Fibrosa Circumscripta, also called **Focal osteitis fibrosa,** is a local condition which may occur in the jaws or other bones as single or multiple lesions, characterised by absorption of bone and its replacement by fibrous tissue. Its etiology is entirely unknown and diagnosis is usually on microscopical evidence and clinical and radiographic evidence taken together, as its histology is identical with that of osteitis fibrosa diffusa. Treatment consists in surgical removal though the lesions occasionally tend to disappear spontaneously, or after hæmorrhage has been produced in them. It affects particularly long bones and the jaws, and occurs chiefly in young people (figs. 961 and 962). It is evident that there is a close relationship between osteitis fibrosa circumscripta and osteoclastoma (giant-celled myeloma) (see p. 958).

Martin Rushton[1] examining an erupted tooth from a male aged 27, which had the surrounding bone typically affected by this disease, figures no aberration from normal in the dentine or cementum and states that the pulp also was normal.

Leontiasis Ossea is a rare condition characterised by a general progressive enlargement of the facial bones and the skull. The enlargement in some cases may be due to osteitis fibrosa and is said sometimes to arise from pyogenic infection ; in other cases it may be a local form of the osteitis deformans of Paget. In these conditions there is absorption of bone with new bone formation, but in osteitis fibrosa (both diffusa and circumscripta) the bone absorption is in excess of bone formation, whereas in the osteitis deformans of Paget there is great formation as well as absorption of bone. Though it is supposed that it may originate from dental infection the ultimate area of bone affected may not be confined to the jaws.

In a case of this nature recorded by G. T. Hankey[2] in a woman aged 43, in poor health and with some loss of weight, there was a gradual painless and diffuse enlargement of the left side of the

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[1] *Guy's Hospital Reports,* 1938, 88, 163.

[2] *Proc. Roy. Soc. Med.,* 1934–5, xxviii, 1676.

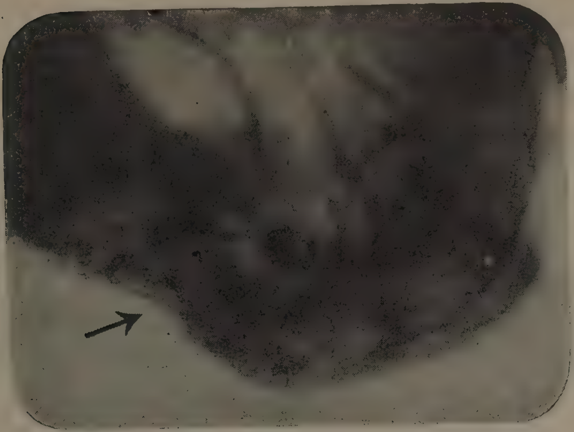

FIG. 963.—Occlusal view of maxilla. (G. T. Hankey.)

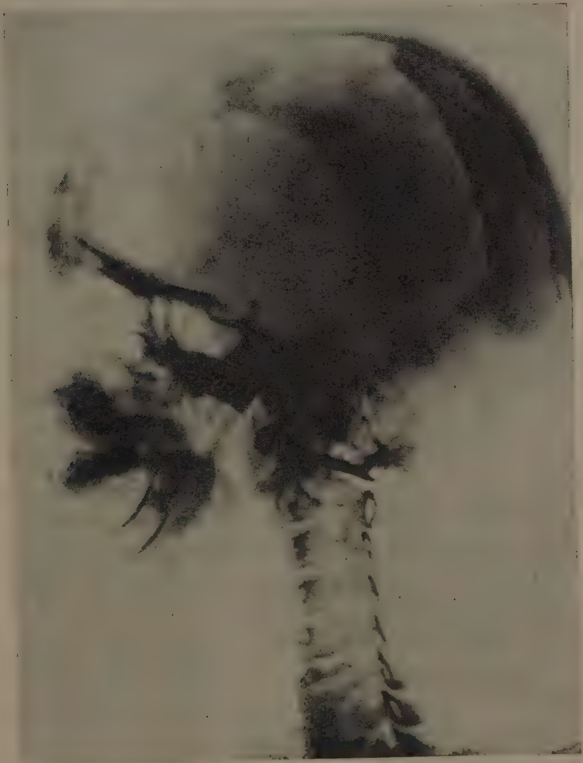

FIG. 964.—Lateral view of cranium. (G. T. Hankey.)

maxilla of nine years' duration following the removal of a maxillary molar. The enlargement eventually spread to the right side, so that the whole maxilla was much enlarged, causing prominence of the upper lip and a generally broad appearance of the face (fig. 963). Seventeen loose and tender teeth affected by chronic parodontal infection were removed, and following this some necrotic bone had to be removed; the base of the maxilla was soft and fibrous and there was no clinical demarcation of the infection. From this time there was a steady improvement in health and no further enlargement of the maxilla. Radiograms originally had shown the cranial bones to be normal, but $2\frac{8}{12}$ years later showed considerable decalcification and mottling of the anterior half of the cranium (fig. 964).

Martin Rushton,[1] examining teeth from a case of osteitis deformans of Paget in a male aged about 70, found the pulps replaced by secondary dentine containing few tubules, acellular calcified material and concentrically laminated islands all calcified into one mass when examined, but admits that this might have been due to age; he also found overgrowth of cementum, gross in one case, though apparently absorption and deposition had alternated in bringing about the change. He considers the cemental changes comparable with those found in bone in this disease.

Lipoid Granulomatosis (Xanthomatosis).—This is a rare condition of the bone of which several cases in the jaws have recently come under notice. In a case under the care of one of us, radiologically (fig. 965) there was a condition of rarefaction of bone which in parts appeared cystic, so that a condition not very dissimilar to a multilocular cyst was seen, but occasionally similarly affected areas appear apart from the main rarefied area, and it does not seem to have a predilection for any particular site. The condition is slowly progressive, loosens overlying teeth and is not confined to any age. Microscopically, masses of eosinophil cells are to be seen and large numbers of connective tissue macrophage cells containing lipoid, which have given rise to the name of the condition. The lesions may occur in any bone (scapula, hip, etc.) or several.

When the disease is adjacent or extends to the stalk of the pituitary a condition of diabetes insipidus may occur and some degree of exophthalmos may be seen. The coincidence of defects in bone produced by the granulomatous condition, with diabetes insipidus and exophthalmos, is sometimes known as the Schüller-

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[1] *Guy's Hospital Reports*, 1938, 88, 163.

Christian syndrome. In cases of doubt a small portion should be removed for biopsy.

Treatment is by means of radium with suitable guards for the protection of adjacent tissues.

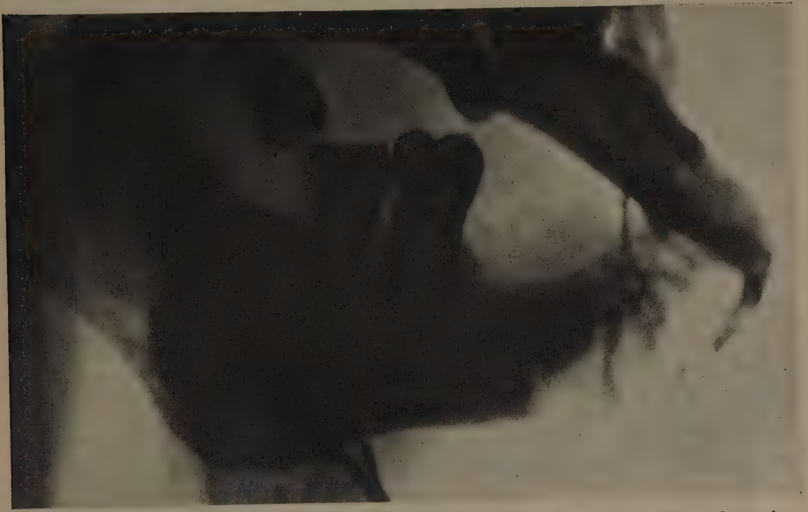

FIG. 965.—Lipoid granulomatosis, in mandible of male aged 36. The first sign of the disease in this case was the loosening of the teeth in the affected area. Diagnosis by biopsy. (E. S. and M. H. Jupe.)

Actinomycosis (Nocardiosis).—*When soft parts are involved* a gradually increasing swelling appears having, at first, a regular even surface with slight superficial hyperæmia ; later this softens in parts and yellowish nodular excrescences appear ; these burst and give exit to a semi-glutinous discharge containing the nodules of actinomycosis. Cicatrisation causes a characteristic puckering, but some sinuses may persist. The swelling is hard and brawny, there is no rise of temperature and usually no pain or enlarged lymphatic glands unless there is secondary infection.

When bone is involved necrosis may be simulated ; the onset is often acute, the gums becoming swollen and bleeding readily, the adjacent teeth loosen and there is considerable swelling of the tissues covering the bone. Eventually, teeth may be lost and portions of bone exfoliated (see fig. 535).

Unerupted misplaced teeth.—These may be, and usually are, teeth of the normal series, in which case their absence from the mouth will be noticed ; or they may be teeth in excess of the normal. The tissues lying over them may, except that they are unduly prominent, be normal in appearance. They give rise to no signs or symptoms, but if they become infected secondary symptoms may occur due to suppuration. Occasionally, misplaced teeth, in endeavouring to erupt, cause absorption of the roots of a tooth already *in situ*, and by pressure on its pulp give rise to considerable neuralgic pain. Radiograms are invaluable for indicating the presence of an unerupted tooth and its position. Certain teeth are more prone to be misplaced than others, their misplacement being either hereditary, congenital or due to lack of room for them in their normal position.

(2) Malignant swellings. Carcinoma.—The important factors in the diagnosis of carcinoma of the jaws are: the age of the individual, which is usually over forty, its relatively rapid rate of growth, its infiltration of surrounding tissues and the involvement of the lymphatic glands which are placed in the course of lymphatic drainage from its area. They are common in males and may occur as malignant papillomata, nodules, or as ulcers. They cause the erosion of bone as they infiltrate it. The disease may commence on the margin of the gum, on the hard palate, the soft palate, on the pillars of the fauces, on the tonsil or the buccal mucous membrane. In the maxilla they sometimes burrow in the substance of the alveolar bone and occupy the antral space, so that there may be but few external signs. They spread via the lymphatics and so give rise to metastases. They are often accompanied by considerable pain.

Sarcoma.—These may occur in the young or in adults; they are vascular, but their vascularity varies and so affects the colour of the growth, they vary in their malignancy and usually grow rapidly and infiltrate surrounding tissues, they spread by the blood-stream and so give rise to metastases in distant parts. Often the veins superficial to them are enlarged.

General Remarks.

There are certain remarks, some of which are applicable to all tumours of the jaws, some particularly to those occurring in the maxilla, and some particularly to malignant growths.

Pressure on nerve trunks may cause pain, and sometimes considerable pain; this latter is particularly true with regard to malignant growths. For instance, the second division of the fifth nerve (superior maxillary) is in close connection with the antrum, entering at the spheno-maxillary fissure, traversing the infra-orbital groove and canal, and opening on the face at the infra-orbital foramen. Implication of this nerve in a growth may give rise to persistent neuralgia before any symptoms of distension of the sinus are apparent. Distension of the antrum may cause expansion of the facial wall, which is probably the least resistant, and so lead to swelling of the face under the lower border of the orbit; the roof of the antrum is the floor of the orbit, and expansion here will give rise to pressure symptoms on the edge and possibly protrusion of the eyeball; and expansion on the nasal aspect may similarly give rise to nasal obstruction. The bony walls of the nasal duct which are partly formed by the maxilla may be pressed on or invaded by growth and so lead to pressure on the duct and consequent epiphora. In cases of malignant disease in the antrum expansion of the facial wall is soon followed by invasion of the overlying skin and the face becomes œdematous.

Transillumination of the antrum is a valuable aid to diagnosis: solid growths, pus, and blood will not transmit light, but other fluid swellings will to a greater or lesser extent; at the same time it must be remembered that the antral cavity is frequently asymmetrical on the two sides. (See pp. 940–941.)

Necrosis of bone may give rise to swelling and tenderness of the gums; this is usually associated with signs of acute inflammation, and though in the later stages, when some destruction has taken place, it is possible to confuse it with malignant disease, in its earlier stages it is not very likely to be mistaken.

Biopsy.—In some cases it is desirable to remove a small portion of the swelling for microscopical examination before a definite diagnosis can be made. A comparison of figs. 712, 950, 951, 954, 961 and 965 will illustrate some of the difficulties in radiological and clinical differential diagnosis. Many, however, believe that in the case of bone tumours the risk of dissemination is so great that the procedure is not justified; a modified procedure which has given satisfactory results is that of *aspiration biopsy*, a hollow needle of moderate size being inserted into the tumour and the contents examined on removal.

Other swellings than those enumerated may also occur, but only exceptionally; such are dermoid cysts in the soft palate and aneurism of the descending palatine artery; but their rarity makes it hardly necessary to discuss their differential diagnosis here; obviously it must depend on the clinical signs present in each case together with a process of exclusion of other causes.

CHAPTER XXXIV

Interference with the Movements of the Temporo-Mandibular Articulation.

Cases of restricted movement of the jaws are not uncommon. The treatment of these cases belongs, as a rule, to the domain of general surgery, but sometimes falls within the province of the dental surgeon. It is necessary, therefore, that the dental practitioner should be familiar with the various diseases which produce this condition. The restriction of movement may be partial or complete.

(A) ETIOLOGY.

(1) Causes connected with the Teeth.—The commonest cause of restricted movement is infection in connection with a partially erupted or misplaced mandibular third molar. In this condition the tooth sac, which is partly open, becomes filled with stagnant matter; infection follows and spreads through the periodontal membrane and thence along the sheaths of the muscles. The inflammatory reaction and exudate leads to impediment to the movement of the muscles.

Infection around the second or first molars as they are erupting, or possibly infection around the maxillary molars, may similarly spread to the soft tissues and cause inability to open the jaws; but as a rule the interference with the movement of the articulation in these cases is not so marked as in the case of the mandibular third molar.

Trismus from irritation of the nerves of the teeth is rare.

Chronic sepsis from the teeth may produce such an enlargement of the neighbouring lymphatic gland as to cause discomfort on movement of the mandible; this is particularly the case with enlargement of the lymphatic gland superficial to the parotid salivary gland.

(2) Trauma.—*Fracture* of the body of the mandible, particularly if there is much displacement or comminution, causes an inability to close the jaws which varies considerably in degree; the degree depends chiefly on the attachment of muscles to the fractured parts, the line of fracture, and the displacement of the part or parts caused by the trauma. *Dislocation*, whether unilateral or bilateral, necessarily prevents normal closure.

(3) Tonsillitis.—In acute tonsillitis the infection may spread to the soft tissues covering the ramus and give rise to pain on opening the mouth. In these cases swallowing causes extreme discomfort; there is little or no swelling over the region of the face, but the tissues behind the angle of the jaw are usually swollen and tender to the touch.

(4) Malignant Disease of the Gums or Fauces.—Malignant disease in the region of the mandibular molars or fauces may lead to interferences with the movement of the mandible. An increasing degree of difficulty in opening the mouth is at times the symptom for which advice is sought, the patient being quite unconscious of the presence of the malignant disease.

(5) Affections of the Parotid Gland.—Swellings of the parotid gland interfere with the free opening of the mouth by causing mechanical obstruction. The commonest cause of such swellings is an inflammation of the gland which may be specific (mumps), or secondary to infection in the mouth, which has spread up the duct. This form of parotitis occurs almost exclusively in adults and most often in females, 85 per cent. according to Morel and 78 per cent. according to Soubeyran and Ribes. It is usually unilateral and, according to Izat,[1] the right side is twice as frequently affected as the left. It is usually associated with abdominal operations and first appears five or six days after operation. It is usually due to an ascending infection of Stenson's duct favoured by lessened salivary secretion. Oral cleanliness before operation is prophylactic. Another cause, but less common, is the presence of a tumour. Swellings connected with the parotid gland are situated below and in front of the ear, and the socia parotidis can usually be felt lying over the masseter. In mumps the condition is usually symmetrical.

(6) Trismus.—By the term 'trismus' is meant spasmodic closure of the jaws; such spasm may be:

(*a*) Tonic.
(*b*) Clonic.

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[1] *Thèse de Paris*, 1935, No. 490.

(*a*) Tonic spasm occurs in tetanus, strychnine poisoning, hydrophobia, hysterical conditions, during anæsthesia and occasionally in cases of fracture of the mandible. In tetanus, spasm of the muscles closing the jaws is usually the earliest of spasmodic symptoms, but in strychnine poisoning the spasms commence in the digits; it is said also to arise reflexly from irritation of the pulps of teeth and the irritation of erupting mandibular third molars, but such cases must be extremely rare.

(*b*) Clonic spasm occurs when the teeth chatter from the stimulus of cold or fear and in hysterical conditions. The term 'trismus' is frequently misused; cases of inability to open the jaws are usually not due to trismus.

(7) Diseases of the Articulation.—In *acute arthritis* there is swelling in the neighbourhood of the joint, the parts are tender to the touch, and any attempt to move the joint causes great pain. In that form of chronic arthritis known as *osteo-arthritis* the movement of the joint is mechanically limited by the formation of new bone around the joint surfaces. In the early stages there is only slight stiffness of the jaw, which is more in evidence in the morning. When the mouth is being opened a clicking sound is audible to the patient and also to the surgeon. As the disease progresses, the stiffness of the jaw increases until, in advanced cases, the articulation becomes practically fixed, owing to alteration in the shapes of the articular surfaces. Osteo-arthritis may be suspected when a patient of forty years or upwards declares a history of gradual loss of power to open the mouth.

In *osseous anchylosis* the jaw is absolutely fixed, but in fibrous anchylosis a slight movement of the jaw can be obtained. There is usually a history of injury or infection of the joint.

In the rare condition known as *hypertrophy of the condyle* there is usually some restriction in the movement of the joint; the enlarged condyle can be felt on palpating the parts over the region of the joint; the chin is pushed to the opposite side and the face is distorted on the affected side.

(8) Cicatrices.—After severe ulceration, scar tissue may be formed to such an extent that the movements of the mandible are considerably restricted, and even complete closure of the jaws may arise from this cause. The scar tissue which forms on the healing of wounds of the elevator muscles of the mandible invariably prevents the mouth from being opened to its fullest extent, the restriction of movement being most marked in the case of injuries to the tendon of the temporal muscle.

(9) Tumours which press upon the jaw from without may occur in the parotid or sub-maxillary regions, or in the situation of the deep cervical glands along the border of the sterno-mastoid muscle. *Deep-seated malignant tumours* may also lead to interference with the mobility of the jaw. Cases of this character are recorded by Coleman, Tomes, and others. This condition may be suspected when, after careful examination, other causes of immobility of the jaw have been eliminated.

(10) In a few cases the immobility of the jaw may be due to such conditions as *exostosis of the zygomatic arch, and ossification of the pterygo-maxillary ligament.*

(B) DIAGNOSIS.

The diagnosis of the causes giving rise to closure of the jaws is best carried out by a process of elimination. The more common causes should be considered first, and these may be taken in the following order: Inflammatory infiltration of the tissues—the most frequent cause being dental, and then, possibly, trauma; mumps; arthritis; mechanical impediment from growths; mechanical impediment from scar tissue; hypertrophy of the condyle; and, lastly, rare conditions such as exostosis of the zygomatic arch.

(C) TREATMENT.

If the trouble is dental in origin an anæsthetic should be administered, the mouth forcibly opened, and the offending tooth or teeth removed. Some difficulty may be experienced in opening the mouth, but a Mason's mouth-gag is generally effective. When the degree of closure at first precludes the use of this instrument, the mouth can usually be forced open by a graduated wedge or spiral cone of boxwood. Instruments for forcibly opening the mouth must be employed with great care. After the extraction of the tooth the socket should be frequently irrigated and the pain relieved by fomentations. Complete recovery of the free movement of the jaw will often rapidly follow treatment, but in severe cases immediate recovery is not to be looked for. Where the third molar is concerned and it is not practicable to remove it, extraction of the second molar may give relief.

If the limitation of movement is due to acute arthritis, wedges should be placed between the molar teeth and a four-tailed bandage applied in order to separate the articulating surfaces and relieve them from pressure. Fomentations should be applied over the region of the condyle. In osteo-arthritis the treatment in the early stages must be constitutional rather than local. The patient should be advised to avoid exposure to the weather as far as possible. For patients who must necessarily be much in the open air a cap with folds to tie over the region of the joint should be recommended. In the more advanced stages, when the jaw has become more or less anchylosed, the operation of excision of the condyle or section through some part of the ramus (Esmarch's operation) may be practised.

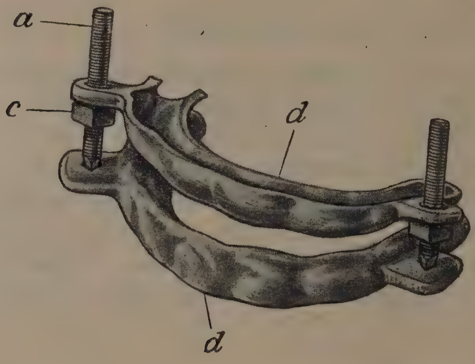

Fig. 966.

Osseous and fibrous anchylosis require for treatment the formation of a new joint ; while for hypertrophy of the condyle excision of the joint may be necessary. If the limitation of movement is due to cicatricial bands, considerable benefit may follow the excision of the scar bands and periodical stretching of the tissues by means of a specially constructed mouth-opener. Fig. 966 illustrates a useful form designed by L. Guanziroli. It consists of shallow metal caps (*d*) covering the surfaces of the maxillary and the mandibular teeth. On either side of the lower cap square-shaped holes are made for the lodgment of the square-shaped tapering ends of the upright bars (*a*) ; by this means some play is obtained at the end of the bar. The upper part of each upright bar is threaded and passes loosely through holes in the upper cap. The apparatus is opened by moving the screws (*c*).

Exostosis of the zygomatic arch and ossification of the pterygo-maxillary ligament are best treated by the formation of a new joint. If the closure is due to external swelling, the treatment consists in seeking for and, if possible, removing the cause of the swelling.

CHAPTER XXXV

Some Affections of the Tongue met with in the Course of Dental Practice.

Certain diseases of the tongue may first come under the notice of the dental surgeon, and it is most important that these diseases should be recognised at once, so that efficient treatment may be given at an early date.

The dental surgeon should know that the shape and appearance of the tongues of different individuals vary considerably within the range of normal, deep congenital fissures are occasionally present and sometimes around the edge may be continued throughout its substance, so as to produce V-shaped indentations. The loss of certain teeth may alter the shape of the resting flaccid tongue, and in course of time such change in outline may be noticeable on protrusion.

(A) IRRITABLE TONGUE.

There is a condition of the tongue when the individual complains of indefinite pain or soreness, or of a pricking, burning sensation. There is no apparent lesion present, though occasionally slight redness may be seen at the part where the symptoms are chiefly noticed. The condition may be a source of considerable mental anxiety, particularly as it is most often noticed about the fifth decade of life, when it is fairly common knowledge malignant disease may show itself.

The cause is not always clear ; in some cases it is due to the sharp edges on teeth which attrition has caused to occur about this age ; in others, recession of gum, sometimes only of slight degree, so alters the environment of the tongue that the new relation of the tongue to the teeth acts as a source of irritation, particularly when there is any gingival sepsis or salivary calculus present ; not only may these mechanical factors act as sources of irritation, but if gingival sepsis is present toxins absorbed via the lymphatics may themselves tend to increase the irritability of the tongue. Other cases occur when some of the teeth are absent and so allow a fidgetty person to chafe his tongue on the teeth adjacent to the spaces ; or when the closing of the bite on account of attrition acts as an irritant by lessening the space available for movement of the tongue, and in either case the irritation may be emphasised when any of the other factors already mentioned is present.

Thick vulcanite dentures, by cramming the tongue into an unaccustomed environment, may also irritate it, and it has been thought by some that vulcanite or the colouring matters used in it may act as irritants. There is, however, little if any evidence that vulcanite or its colouring matters can so act, though it is possible that the growth of organisms on the rougher surfaces, which are adapted to the soft tissues, may act as an irritant. In any case a struck metal denture, being thinner and unable to harbour micro-organisms, should be substituted, and this frequently affords relief. Other treatment will depend on the factors present and consists of the removal of sharp edges caused by attrition and polishing the newly exposed surface, the removal of all calculus, salivary or serumal, and the polishing of the underlying surfaces and the filling up of any spaces occasioned through loss of teeth by the fitting of a denture. It is important to assure the patient that there are no signs of malignant disease, but it is not always easy.

Another and rather different source of irritation sometimes occurs, and often at an earlier age ; this is caused by the enlargement of the *foliate papillæ* at the sides of the posterior portion of the tongue, and is usually caused by the absorption of toxins from sepsis of dental origin. Treatment as far as the dental surgeon is concerned consists in the removal of all possible infection of dental origin, and, should the condition not then clear up, referring the patient to his medical practitioner for the treatment of other possible sources of infection.

(B) PERNICIOUS ANÆMIA.

A sore tongue is an early, often the first, symptom in pernicious anæmia, and in all cases where local irritations and syphilis can be excluded this must be regarded as a possibility ; such symptoms are stinging or biting sensations, usually along one side and radiating towards the lip. The normal coating may disappear, leaving a pale, smooth and glossy or raw, tender surface (' beefy tongue '); sometimes there is a true geographical tongue. These appearances may be noted in almost every case and are so constant that they may be considered as one of the most important signs of pernicious anæmia. Other symptoms follow at a greater or less interval of time; such cases, beyond demanding the attainment of a high degree of oral hygiene at the hands of the dental surgeon, should be immediately passed over to a medical practitioner for appropriate treatment. (See also ' Geographical Tongue.')

(C) LEUCOPLAKIA.

There are several conditions which may give rise to an appearance of leucoplakia on the tongue or mucous membrane of the mouth. The most important of these is that known as **Chronic Superficial Glossitis.**

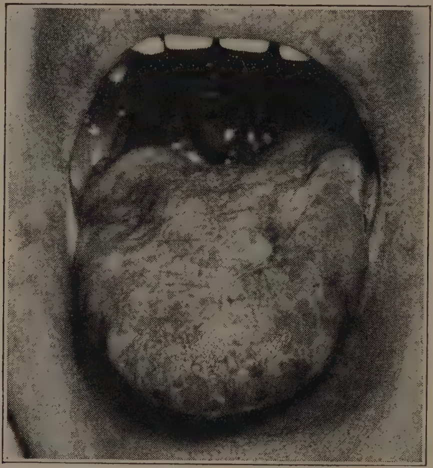

FIG. 967.—Leukoplakia of tongue associated with syphilis. (From *Journ. Amer. Dent. Assoc.*)

The signs are most often seen on the papillated portion of the tongue and are due to an inflammatory condition of the mucous and submucous tissues accompanied by changes in the surface epithelium. There is usually a history of long-continued irritation such as may be caused by excessive smoking, strong spirits, or the habitual use of strong condiments; there is very frequently a history of syphilitic infection of long standing, but the complaint is but seldom influenced by any antisyphilitic remedies ; there is usually chronic dental sepsis (fig. 967).

The disease does not affect the tongue uniformly but occurs in patches. The earliest signs are the enlargement and heaping up of the filiform papillæ, which present a reddened appearance ; later, as the epithelium on them becomes thickened, they present a sodden yellowish or dirty white appearance. Later on these hypertrophied papillæ become rubbed off and a smooth glazed and reddened area is exposed, which may later assume a pearly bluish-white appearance, slightly depressed below the adjacent papillated area of the tongue ; all these appearances may be present at the same time. Abrasions of the epithelial surface occur and, in conjunction with infection and other irritants, give rise to fissures which cause considerable pain, and it is frequently this symptom which first brings the patient for treatment. Under treatment, fissures will often rapidly heal, but they frequently recur. When the hypertrophy of papillæ is extreme the name *ichthyosis* is applied to the condition. The condition is especially important because it is so frequently a precancerous condition, over 30 per cent. of these cases ultimately resulting in carcinoma linguæ. The treatment of the condition falls to the medical practitioner, but the dental surgeon can do a good deal. Dental treatment consists in removing all possible sources of infection and irritation of dental origin ; the mouths of persons suffering from this disease frequently show much oral sepsis, occasioned usually by untreated and often advanced parodontal disease ; in such cases, owing to the seriousness of the condition, treatment should be radical rather than conservative.

Other conditions which may also give rise to an appearance of leucoplakia are :—

Lichen Planus.—In this condition the patches are mainly confined to the mucous membrane lining the cheeks rather than the tongue. The lesions are not so thick or white as are those of syphilitic leucoplakia, but more usually bluish-white (fig. 968). The skin lesion typical of the condition and consisting of irritating flat-topped burnished papules may also be present on the flexor surface of the wrist or the shins or elsewhere.

Herpes Recurrens.—In this condition patches may appear on the mucous membrane of the cheeks or on the tongue ; similar lesions may appear concurrently on the glans penis.

Lupus Erythematosus.—In this condition the leucoplakial patches occur on the buccal mucous membrane rather than on the tongue and have a reddened border, a condition not seen in syphilitic leucoplakia; and there is usually present the scaly fixed erythema, leaving thin scars often in the central portion of the face in typical butterfly pattern. It is not due to the tubercle bacillus.

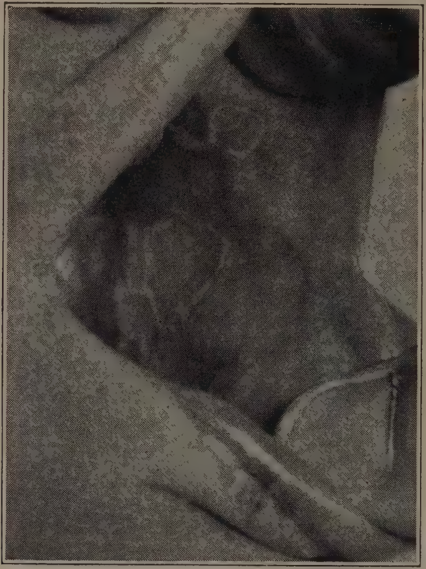

FIG. 968.—Lichen planus of buccal mucosa.
(From *Journ. Amer. Dent. Assoc.*)

Thrush.—When this disease occurs in adults, when it is known as Monilil, it may give rise to white patches on the tongue.

(D) GEOGRAPHICAL TONGUE.

This is a form of superficial glossitis which results in a superficial epithelial erosion and shows as a smooth moist surface having the edge slightly raised and opaque and irregular in outline; it is one of the forms of glossitis which occurs as an early sign of pernicious anæmia.

Other lingual conditions which should be known to the dental surgeon are :—

(E) BLACK HAIRY TONGUE.

This condition is due to the growth of a yeast-like fungus, *Cryptococcus linguæ pilosæ*, which occurs in patches, sometimes extensive, on the anterior two-thirds of the dorsum of the tongue ; the patches vary in colour from pale brown to black and the hair-like filaments are elastic and up to three-fourths of an inch in length. On the removal of the mass by scraping, the underlying papillæ may appear normal even when the patch has been of long standing, though the filiform papillæ have been found hypertrophied in some cases. The hair-like filaments lie in the direction in which they are pushed or stroked, and are said by Gleave and Hallam [1] to consist of ‘ elongated masses of epithelial cells and scales ’ which are composed of ‘ more or less keratinised epithelium attached to the summits of the filiform papillæ ’ and to be ‘ probably a great exaggeration of the thick fur often seen in disease.’ They found that the colour was due to a diffuse brown coloration of these epithelial cells, and that the filaments were covered on all sides with an enormous number of the yeast cells ; on laboratory culture of the yeast they found no pigment was produced, but others have found laboratory cultures to show a brown colour. The fungus seems only to flourish in an acid environment.

Gleave and Hallam found that the use of an alkaline mouthwash (liquor potassæ), by making the environment unfavourable for the growth of the fungus, rapidly cured their case, but record that in other cases ‘ the lesion is said to disappear and recur in spite of treatment.’ For such cases Liq. arsenicalis, 1 drachm to the ounce used as a paint three times daily has been found satisfactory, as also has the application of 50 per cent. trichloracetic acid after drying the tongue, one application of this latter usually being sufficient. Cases have also been cured by the internal administration of potassium chlorate, 5 grains in water thrice daily for ten days, and then after a lapse of a fortnight resumed for a further ten days. Any oral sepsis present should of course be eliminated. The disease is rare in England, but apparently rather commoner in France, and has been mistaken for melanotic sarcoma. In addition to the signs already mentioned a burning sensation may be felt in the tongue or, when it extends far back, an irritation accompanied by cough. Apparently the backward direction of the filaments on to the posterior third of the tongue has given rise to the statement that the fungus may also grow behind the circumvallate papillæ.

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[1] *Brit. Med. Journ.*, April 1931.

(F) STREPTOCOCCAL INFECTION.

In their description of streptococcal fever which includes streptococcal bacteriæmia and septicæmia, Ryle and Smith[1] emphasise the tongue condition as being one of the four clinical manifestations of this disease when due to a hæmolytic strain of the organism. The tongue is described as being smooth, red, desquamated, and sore ; in addition, there is also a rapidly progressive anæmia, diarrhœa, and albuminuria with red blood-cells and casts. According to A. Bulleid it is associated with 'cracking' at the angles of the mouth.

(G) ULCERATIONS OF THE TONGUE.

(1) Simple. (*a*) *Dental.*—This is the commonest type of ulcer of the tongue. The predisposing cause is the sharp edge of a tooth, or the rough edge of a denture. In some cases the ulceration is quite superficial, but, nevertheless, is very painful when speaking or eating. The surface is usually surrounded by a zone of inflammation. In other cases the ulcer extends deeper, the edges being irregular and abrupt, the surrounding mucous membrane inflamed, and the floor of the ulcer bathed with pus. The lymphatic glands may become enlarged from sepsis. When dental ulcers have existed for some considerable time the edges and base may become indurated, and, in appearance, the condition may mimic carcinoma. Not infrequently a carcinoma may develop at the site of a simple ulcer. If there is any doubt about the character of the ulcer its edge should be submitted to microscopy.

Treatment consists in removing the cause. Rapid improvement and healing generally follow, but if this does not occur a V-shaped portion of the tongue containing the ulcer should be excised.

(*β*) *Injuries from other Causes than the Teeth.*—Ulcers may arise from burns, bites, stings, etc. The history of the case gives the clue to diagnosis.

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[1] *Guy's Hospital Reports,* 1931.

Treatment.—An antiseptic mouth-wash is usually all that is needed.

(γ) *Dyspeptic* ulceration is most frequently met with near the tip of the tongue on the dorsal aspect. The ulcers are generally multiple and superficial, the portion of tongue around them being nearly always slightly inflamed, while symptoms of dyspepsia are present.

Treatment consists in attending to the dyspepsia, employing chlorate of potash or other mild antiseptic mouth-washes. Silver nitrate should never be applied to the tongue.

(2) Infective. (α) *Syphilis.*—(i) *A primary chancre* is usually situated on the tip or the edge near the tip. It is characterised by the rapid formation of a hard nodule with superficial ulceration. The submaxillary glands are enlarged, and secondary symptoms of syphilis soon appear.

(ii) *Secondary.*—In the second stage of syphilis mucous tubercles may appear on the tongue. These tubercles consist of patches of enlarged and infiltrated papillæ, the superficial epithelium becomes soddened and gives rise to a characteristic yellow-white appearance. The surface of the mucous tubercles is liable to be injured, and ulceration of a septic type appears. The ulcer is therefore septic in character grafted on a syphilitic lesion.

(iii) *Tertiary.*—(*a*) *Deep intramuscular gumma* forming a nodule in the substance of the dorsum of the tongue. The swelling, which is painless, breaks down and forms a deep excavated ulcer. (i) The edges are sharply cut ; (ii) the induration of the surrounding tissue rapidly disappears under treatment ; (iii) the floor may present a wet washleather-like appearance in the early stages before the slough has disappeared ; (iv) the situation of the ulceration is usually the dorsum of the tongue immediately to one side of the median line ; (v) the submaxillary lymphatic glands are not as a rule enlarged ; (vi) profuse salivation is not present ; (vii) there may be pain, but not so severe as in epithelioma.

(*b*) *Multiple superficial gummata.*—These break down and form superficial ulcers.

(*c*) *Fissures and ulcerations* in connection with chronic superficial glossitis.

(β) *Tubercle.*—Tuberculous ulceration of the tongue is generally secondary to tuberculosis of the lungs or larynx and is due to a direct infection from the sputum. The common situation is the under-surface of the tip of the tongue. The ulcers are superficial, often multiple and very painful. A zone of inflammation is present around the ulcer, and, occasionally, caseation of the floor can be seen. The coincidence of the ulcers with well-marked symptoms of tuberculosis of the lungs or larynx is sufficient to prevent the condition being mistaken for either syphilis or carcinoma. Occasionally, the clinical symptoms of the primary disease are not very obvious.

(γ) *Actinomycosis.*—Ulceration from this cause is rare. A hard, brawny swelling of the tongue forms and breaks down, giving vent to a gelatinous-like discharge containing the characteristic granules. More usually there is a hard, woody swelling of the tongue accompanied later by multiple sinuses from which matter containing yellowish granules discharges, which are found microscopically to contain the *Leptothrix actinomycosis.*

(3) Malignant. (α) *Carcinoma.*—This most serious disease of the tongue may start :—

(i) In chronic ulcers or fissures, dental in origin, and very commonly situated at the borders of the tongue in the molar and premolar region.

(ii) As a warty outgrowth, with infiltration of the base and surrounding tissues.

(iii) As a nodule.

(iv) In association with chronic glossitis originating in an ulcer, fissure, or ichthyosis, or appearing as a diffuse infiltration.

The characteristics of a carcinomatous ulcer are as follows: (i) Raised, sinuous, hard and everted edges ; (ii) the surrounding mucous membrane is indurated ; (iii) the floor irregular, in part excavated, and in part showing warty prominences and covered with a foul discharge ; (iv) the base indurated, and in the latter stage, the tongue fixed ; (v) a darting pain ; (vi) profuse salivation ; (vii) enlargement of the neighbouring lymphatic glands (not in the very early stages) ; (viii) interference with speech and mastication. The disease is more prevalent in men than in women, and occurs generally in those past the age of forty.

In the early stages there may be no pain. When carcinoma occurs in the anterior two-thirds of the tongue there is frequently an accompanying history of syphilis, especially in those cases commencing as chronic superficial glossitis, but in cases which occur in the posterior third syphilitic infection is not often an accompanying factor.

Ministry of Health statistics show that the incidence of this disease varies considerably in different occupations, being greatest in potmen and smallest in nonconformist ministers.

Treatment.—The dental surgeon is concerned in (1) referring the patient to the general surgeon for radium, diathermy, or such other operative treatment as may be deemed necessary, and (2) preparing the mouth for such treatment by removing all possible sources of irritation of dental origin whether mechanical or toxic.

As the mouths of patients suffering from this complaint are usually in a highly insanitary state, treatment should be radical rather than conservative, and must be rapid, as the progress of the untreated disease is rapid and the prognosis is very bad in cases where treatment is not commenced till the disease is advanced.

Early recognition of the disease is of the utmost importance. The presence of a wart on the side, tip, or, indeed, any part of the tongue in a person over thirty-five years of age should always be regarded with suspicion, and the patient *at once* advised to seek skilled assistance. All ulcers of some standing should be submitted to a careful examination. The particular points to which attention should be directed are: (i) the history of the ulcer; (ii) the character of the edges; (iii) the condition of the surrounding mucous membrane.

Ulceration in patients over forty is always serious, and in doubtful cases it is better to have the suspicious portion excised.

(β) *Rodent ulceration* is extremely rare, and scarcely needs description here, and the same may be said of (γ) *sarcoma*.

CHAPTER XXXVI

Local Anæsthesia in Dental Surgery.

BY THE LATE F. ST. J. STEADMAN, L.R.C.P., M.R.C.S., L.D.S., D.P.H.

CERTAIN drugs when injected have the power of producing a temporary paralysis of nerve fibres, which blocks the passage of impulses to the brain. The drug employed may be injected at the site of operation (submucous anæsthesia) or in the neighbourhood of the nerve trunks at some distance from the field of operation (regional or block anæsthesia).

To obtain satisfactory results a good syringe is essential. It should consist of a stout metal or glass barrel of convenient size (holding at least two cubic centimetres) and a washerless vulcanite plunger with a stop, which prevents its being withdrawn altogether from the barrel with an unpleasant jerk. The syringe should be capable of being taken to pieces and boiled if required, although it will deteriorate and will not work smoothly if boiled too frequently. The syringe is best kept in a large glass jar, which should be of sufficient size to immerse it completely.

If novocain is the drug employed, the following fluid advocated by A. E. Smith[1] (but in which the strength of the Phenol has been doubled) will be found to keep the syringe in excellent condition, sterile and ready for immediate use :—

	Parts.
Phenol	40
Sodium biborate	5
Glycerin	200
Distilled water q.s. ad. Misc.	1,500

It is good practice to wash out this fluid by passing a small quantity of cold boiled water through the syringe just before drawing up the injecting solution.

The plunger may be lubricated with a very small quantity of carbolised vaseline; but not too much should be used or the needle will become blocked. As short nozzles as possible should be used, for if the needle breaks it will do so probably at the weakest point, *i.e.* just where it emerges from the hub. So that if the operator makes it a rule never to insert the needle into the tissues up to the hub, when a break occurs a short piece will always be left protruding, allowing the broken fragment to be rapidly and easily removed.

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[1] 'Block Anæsthesia and Allied Subjects,' A. E. Smith.

The manufacture of irido-platinum needles at present leaves much to be desired, as they are expensive and break readily at the point of junction with the hub. Steel needles are cheaper and can be discarded after being used a few times.

There are two lengths of rustless steel needles, viz. forty-two and twenty-one millimetres. The long needles are required for the regional and the short for the submucous injections.

An angle attachment to the syringe is necessary for the maxillary molar injections.

Novocain.—The drug now most commonly employed is novocain (para-amino-benzoyl-diethyl-aminethanol hydrochloride). Cocaine of recent years has been almost entirely discarded, as it is a protoplasmic poison, is uncertain in its action, and is decomposed by boiling—a very great disadvantage. Novocain is a white crystalline powder capable of being heated to 120° C. without decomposition. It may be synthetically prepared, so that its action is reliable and uniform. It is soluble in water (one part) and in alcohol (thirty parts). It keeps for several months without decomposition. It is seven times less toxic than cocaine. Its toxicity is so relatively light that 24 c.c., or twelve syringes full of a 2 per cent. solution, may be employed, a quantity that is very seldom, if ever, necessary for dental purposes. It is incompatible with lysol and alkaline carbonates. It possesses a great advantage in that it is not a habit-forming drug. When injected in an isotonic solution it does not produce irritation of the tissues, but the patient may notice a slight burning sensation for a few minutes after the injection, which is due to dilatation of the capillaries. It is a powerful local anæsthetic, producing an anæsthesia which is little inferior to that produced by cocaine, although its action is slower.

Suprarenin (Adrenalin), the active principle of the suprarenal gland, can now also be prepared synthetically. It can be sterilised by boiling, but is liable to decompose if the boiling is prolonged. The solution sometimes turns a pale pink as it is being boiled, due to a slight oxidation of the drug taking place; the solution may, nevertheless, be safely used, provided the discoloration does not pass from a pale pink through red to brown. Suprarenin will keep for many months either alone or in tablet form with novocain, but not if calcium chloride is added. It is decomposed by alkalies. It is a powerful vaso-constricting agent, and for this reason prevents the rapid absorption of the local anæsthetic, so that not only is the duration of the anæsthesia much prolonged, but a larger quantity of the anæsthetising drug may be employed without danger of toxic effects. Suprarenin is, however, itself highly toxic, and is the cause of the palpitation and slight distress which some patients experience a few hours after the injection. It is probably also responsible for the alarming symptoms of collapse which have been recorded in certain cases and wrongfully attributed to the toxic effects of novocain. It is contra-indicated in organic heart disease.

Compressed tablets of novocain either alone or in combination with suprarenin are manufactured by the Saccharin Corporation Co., Ltd. The 'F' tablets consist of 0·05 grm. ($\frac{3}{4}$ gr. approximately) of pure novocain and 'E' tablets of 0·02 grm. ($\frac{1}{3}$ gr. approximately) of novocain and 0·00005 grm. ($\frac{1}{1200}$ gr. approximately) of suprarenin. It is most important to distinguish between these two tablets, for whereas it is permissible to use seven or eight, or even more of the 'F,' not more than two 'E' tablets should be used, otherwise there is a slight danger of toxic symptoms due to an overdose of suprarenin supervening, manifested by profuse perspiration, dilatation of the pupils, pallor, laboured respiration and palpitation of the heart, followed by collapse.

It is important to inject these drugs in a solution, isotonic with blood, *i.e.* a solution containing salts of the same character and as nearly as possible in the same proportion of those in the blood. For the tissue cells are surrounded by a membrane through which water, but not salts, may penetrate by osmosis. If a hypertonic solution (a solution too rich in salts) is injected, the tissue cells become contracted and crenated; and if a hypotonic solution (poor in salts) is injected, the cells swell up and may actually rupture; both these conditions are a probable cause of after-pain.

To obtain the best results, it is advisable to prepare a fresh quantity of distilled water daily. Two ounces per diem will usually be sufficient for most operators. To this amount a 'Ringer' tablet composed as follows is added, making approximately an isotonic solution [1] :—

Calcium chloride	0·011 grm.
Potassium chloride	0·011 ,,
Sodium chloride	0·51 ,,

Two c.c. of this solution are boiled for a couple of minutes and one 'F' and one 'E' tablet added, and the whole boiled again for another minute. The solution is allowed to cool slightly and is then ready for injection.

This daily distillation may be a little troublesome, but it is most important to be able to rely absolutely upon the purity of the injecting solution. Moreover, it is probable that the use of stale distilled water is one of the causes of after-pain.

In recent years various chemists have placed on the market certain proprietary solutions which are satisfactory and may be safely used. It is, however, important to use only solutions of which the manufacturers are prepared to give the full formula. It is most unwise to inject a solution the exact content of which is unknown to the operator.

Many of these solutions have the disadvantage that they are not very stable, in so far as they become more acid as they are kept, and a too acid solution is liable to give rise to after-pain.

The Pharmaceutical Manufacturing Co., Ltd., have now overcome this difficulty by putting up their solution of Novutox in two bottles, that is to say with the alkalising fluid separate. The formula is as follows :—

Novutox—D.S. 3.

Potassium chloride	2·4 grms.
Calcium chloride	3·0 ,,
Sodium chloride	24·0 ,,
Acid Benzoic	20·0 ,,
Procaine hydrochloride	600·0 ,,
Capryl-hydrocupreinotoxin HCl	4·0 ,,
Adrenalin	0·4 ,,
Sodium thiosulphate	14·4 ,,
Thymol	4·0 ,,
Alcohol 95 per cent...	50 c.c.
Glycerine	50 c.c.
Distilled water to	10 litres

This solution when mixed with an equal quantity of the alkalising solution as follows :—

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[1] These tablets are made by Messrs. Wright, Layman and Umney, Ltd., and may be obtained through any chemist.

Novutox Alkalising Solution.

Sodium salt para-chlor-meta-cresol ...	10 grms.
Sodium carbonate	12·34 ,,
Sodium bicarbonate	56·43 ,,
Sodium metabisulphite	3·52 ,,
Distilled water to	10 litres

gives a 3 per cent. solution of the pH value of 7·4.

They also prepare a solution of cocaine in the following formula :—

Isooctylhydrocupreinotoxin hydrochloride ...	0·002
Benzoic acid	0·200
Adrenalin	0·00125
Thymol	0·040
Cocaine hydrochloride	0·950
In Ringer's solution to	100·000

These solutions contain sufficient antiseptics to keep them sterile. They contain a small percentage of adrenalin. Experience has shown that in many of these proprietary solutions the percentage of adrenalin has been too high up to the present.

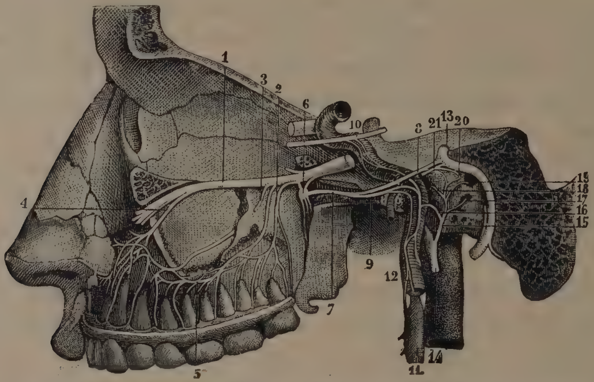


FIG. 969.—Deep view of the maxillary nerve and the spheno-palatine ganglion. 1, Maxillary nerve ; 2, posterior superior dental ; 3, middle superior dental ; 4, anterior superior dental ; 5, superior dental plexus ; 6, spheno-palatine ganglion ; 7, Vidian nerve ; 8, great superficial petrosal ; 9, great deep petrosal ; 10, a part of the sixth nerve, receiving twigs from the carotid plexus of the sympathetic ; 11, superior cervical sympathetic ganglion ; 12, its ascending branch ; 13, facial nerve ; 14, glossopharyngeal nerve ; 15, its tympanic branch ; 16, carotico-tympanic twig joining the sympathetic ; 17, filament to the fenestra cochleæ ; 18, filament to the Eustachian tube ; 19, filament to the fenestra vestibuli ; 20, small superficial petrosal. (From Sappy, after Hirschfeld and Leveille. Quain's 'Anatomy.')

In the latter solution, containing the cocaine, the drug is in a weak solution, and the addition of the adrenalin limiting absorption into the tissues, renders this a safe solution to inject.

The mucous membrane through which the needle is to pass should be dried and painted with tincture of iodine. Great care should be taken that the area so treated should not be again contaminated by the movement of the patient's tongue before the injection is made, and also that during the injection no part of the patient's lips or tongue should be allowed to touch the needle point. If such an accident occurs, the needle must be withdrawn immediately and re-sterilised before proceeding with the injection.

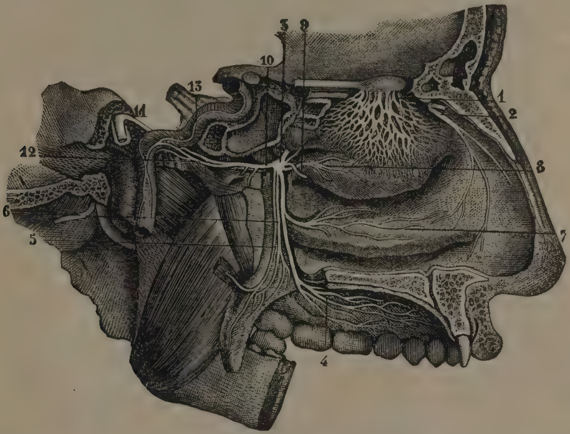


Fig. 970.—Nerves of the nose and Meckel's ganglion. 1, Network of the external branches of the olfactory nerve; the area of distribution is not so large as represented here, and its fibres do not join to form a network; 2, naso-ciliary nerve, giving its external branch to the outer wall of the nose; the septal branch is cut short; 3, spheno-palatine ganglion; 4, ramification of the large palatine nerve; 5, small, and 6, external palatine nerve; 7, inferior nasal branch; 8, superior nasal branch; 9, naso-palatine nerve cut short; 10, Vidian nerve; 11, great superficial petrosal nerve; 12, great deep petrosal nerve; 13, the sympathetic nerves ascending on the internal carotid artery. (From Quain's 'Anatomy.')

While a good knowledge of the fifth cranial nerve is necessary if the best results are to be obtained, a detailed anatomical description would be out of place in a work of this kind. However, a few essential points may be mentioned. The maxillary teeth are supplied by the second division of the fifth cranial nerve, which leaves the skull through the foramen rotundum, crosses the spheno-maxillary fossa to enter the infra-orbital canal, along which it passes to emerge upon the face through the infra-orbital foramen, where it terminates in three sets of branches: the palpebral, the nasal, and the labial (fig. 969). In the spheno-maxillary fossa the nerve gives off the superior dental branch, which descends upon the zygomatic surface of the maxilla, until, about half-way down, it pierces the outer surface of the bone to supply the third, second, and frequently the posterior buccal root of the first molar teeth. In the infra-orbital canal the middle superior dental branch passes along a canal in the outer wall of the maxillary sinus to supply the first molar and the premolar teeth.

The canine and incisor teeth are supplied by the anterior superior dental branch, which leaves the main trunk about five millimetres before it emerges upon the face. These three nerves anastomose freely with one another, forming a plexus, and, with the corresponding plexus of the opposite side, form an outer nerve loop, which supplies the outer alveolar plate and overlying gum in addition to the teeth.

The hard and soft palate, the inner alveolar plate and the overlying gum are supplied by an inner nerve loop formed by the anterior palatine and naso-palatine branches of Meckel's ganglion (fig. 970).

The mandibular teeth are supplied by the inferior dental nerves, which, by anastomosing with each other, form an outer loop to supply the outer alveolar plate and overlying gum. The lingual nerves forming an inner loop likewise supply the inner alveolar plate and gum (fig. 971). Another branch of the fifth also requires notice—namely, the long buccal nerve which supplies the inner side of the cheek and gum on the outer side of the mandibular molar teeth (fig. 971). For extraction of teeth, the deep scaling of sub-gingival tartar, or the removal of cysts, it is necessary to block both loops: whereas for conservative work such as the excavation of sensitive cavities, the extirpation of pulp, or apicectomy, the outer nerve loop only need be blocked.

If the outer and inner bony surfaces of the maxilla be examined, it will be seen that numerous canaliculi perforate the compact layers just above the apices of the roots of the teeth. In the mandible it will be seen that, except for a few canaliculi below the level of the roots of the canine and incisor teeth on the outside, and in the neighbourhood of the genial tubercles on the inside, these canaliculi are few in number. This is an important practical point, as these canaliculi greatly assist the infiltration of the anæsthetising fluid.

Technique.—In extensive operations in the maxilla, such as the extraction of several or all of the teeth, or the removal of tumours or cysts, where both the outer and inner nerve loops are involved, the simplest method is to block the second division of the fifth cranial nerve as it crosses the spheno-maxillary fossa. Such an injection will catch not only all three of the nerves supplying the teeth and the adjacent parts, viz. the posterior, middle, and anterior superior dental nerves, but also Meckel's ganglion, from which is formed the inner nerve loop. If all the upper teeth are to be removed, this injection must be done on both sides, but if the teeth from one half of the jaw only, then one injection in the spheno-maxillary fossa will suffice,

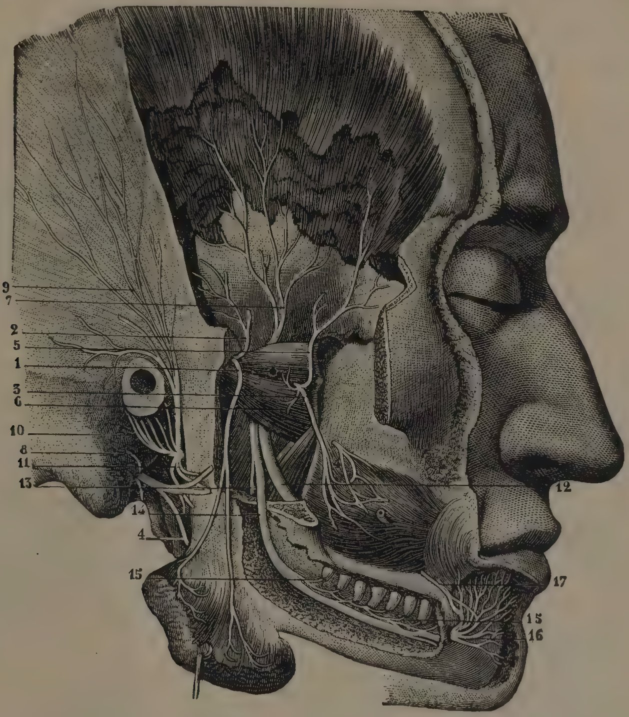


Fig. 971.—View of the branches of the mandibular nerve from the outer side. The zygoma and part of the ramus of the lower jaw have been removed; the dental canal has been opened up; the lower part of the temporal muscle has been taken away, and the masseter muscle turned down. 1, Masseteric nerve; 2, posterior deep temporal nerve; 3, buccal nerve; 4, branch of the facial; 5, anterior deep temporal nerve; 6, filaments given by the buccal to the external pterygoid muscle; 7, middle deep temporal nerve; 8, auriculo-temporal nerve; 9, its temporal branches; 10, its branches to the meatus and auricle; 11, its union with the facial; 12, lingual nerve; 13, mylohyoid nerve; 14, inferior dental nerve; 15, its twigs to the teeth; 16, mental branch; 17, branch of the facial uniting with the mental. (From Quain's 'Anatomy.')

provided that the overlapping branches from the anterior superior dental and the naso-palatine nerves of the opposite side be anæsthetised.

The distance between the reflection of the mucous membrane from the maxillary tuberosity and the nerve trunk itself is from twenty-seven to thirty-three millimetres (Smith); therefore, in accordance with the rule already laid down that the needle should never be inserted up to its junction with the nozzle, a long needle is required. One may use either a right angle irido-platinum forty-millimetre needle, or a long rustless steel needle joined to the syringe by the angle attachment. The point of insertion of the needle is behind the tuberosity of the maxilla at the highest point of the reflection of the mucous membrane of the cheek from the bone, that is to say, above and to the buccal aspects of the roots of the third molar. The point of insertion should be well away from the gum margin, and the needle should be advanced in an upward, inward, and slightly

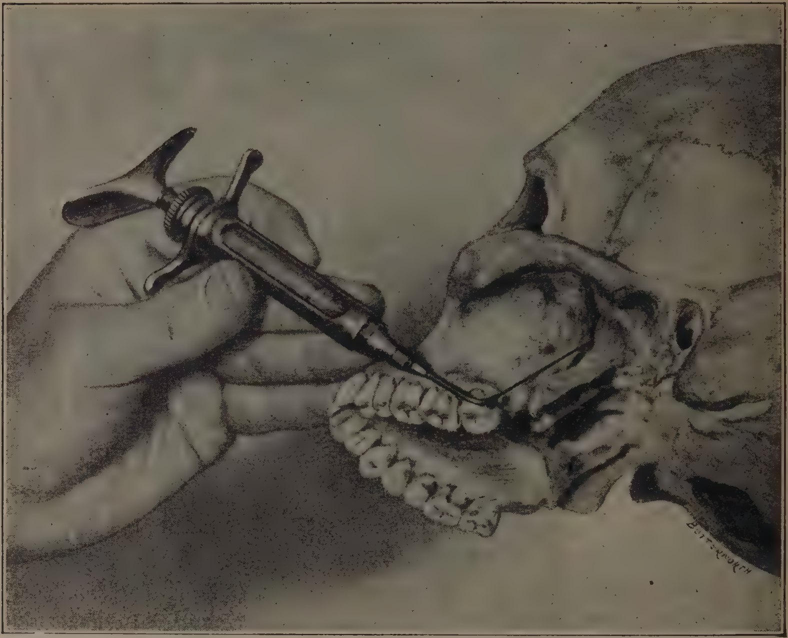


FIG. 972.—Blocking the maxillary division in the spheno-maxillary fossa. (From Steadman's 'Local Anæsthesia in Dental Surgery.' Henry Kimpton.)

backward direction for a distance of about thirty millimetres. If the direction is correct the needle will slip in easily and painlessly (fig. 972). If, however, the direction is too far backwards, the point of the needle will strike against the greater wing of the sphenoid and its further passage will be prevented. Should this happen, it is necessary to withdraw the needle and change its direction. On the other hand, should the needle point be directed too far forward, it will be caught up in the periosteum overlying the maxillary tuberosity. Two cubic centimetres of a 2 per cent. solution of novocain should be slowly injected. Occasionally, at the moment of the commencement of the injection, there is a transitory blanching of the area of the skin of the face which is supplied by the second division, owing to the contraction of the corresponding arteries. A profound anæsthesia extending over the cheek as high up as the lower eyelid, as far forward as the inner side of the nose, downwards to the upper lip, and backwards along the cheek in a line with the mouth, will develop in from about twenty-five to thirty-five minutes. If this injection is made on one side only, the three molars and two premolar teeth may be safely removed. If it is intended to remove the canine and incisor teeth also, it will be necessary to block the overlapping branches of the anterior superior dental nerve of the opposite side by a submucous infiltration over the apex of the canine, and an injection in the palate behind the second incisor at about the level of its apex. Occasionally the patient experiences a slight twitching sensation in the eye, followed by diplopia, which usually passes off in about twenty minutes. Should this happen, care should be taken to explain to the patient that there is no cause for alarm.

For operations upon the second and third maxillary molar teeth it is only necessary to block the posterior superior dental nerve, which can easily be reached as it enters the canal about half-way up the posterior surface of the zygoma. For this injection a short needle and the angle attachment previously mentioned should be used. The point of insertion of the needle is behind the root of the zygoma at about the level of the apex of the posterior buccal root of the second molar. The needle is advanced in an upward, backward, and inward direction through some of the fibres of the buccinator and external pterygoid muscles, care being taken to keep fairly close to the bone, but not close enough to pass under the periosteum. This injection is painless and simple. If a straight attachment to the syringe is used there is a risk of pushing the needle backwards into, and even through, the fibres of the external pterygoid muscle, behind which is the pterygoid plexus of veins, and not only missing the nerve altogether but causing a hæmatoma.

Anæsthesia develops in from three to five minutes, when cavities in these two teeth may be prepared, or their pulps extirpated. If extraction is required, the anterior-palatine nerve must be anæsthetised by a palatal injection, the needle being inserted exactly half-way between the mid-line and the margin of the gum a little behind the root of the third molar.

For operations upon the maxillary first molar the same injections are required. In addition to these injections, however, a sub-mucous, sub-periosteal injection should be made over the apex of the anterior buccal root to anæsthetise the middle superior dental nerve which supplies it.

For operations upon the premolars, the sub-mucous and sub-periosteal injection above the level of their apices will generally suffice for conservative work, with an additional palatal injection: also above the apices of the roots for extraction.

For operations upon the canine and incisors, either an infra-orbital regional or a sub-mucous, sub-periosteal injection above their apices may be employed. For the infra-orbital injection a long straight needle with a short nozzle is used. The infra-orbital foramen is first palpated. It will be felt a finger's-breadth below the junction of the inner and outer two-thirds of the infra-orbital margin. With the pulp of the forefinger over this spot the lip is held outwards between this finger and thumb, and the needle is inserted into the mucous membrane, in the inner side of the cheek opposite the second premolar, two or three millimetres from the point of reflection of the mucous membrane from the bone. It is now advanced along the direction of this root directly to the infra-orbital foramen. The fluid is then deposited under the pulp of the forefinger and massaged in an upward and backward direction towards the infra-orbital canal (fig. 973). Anæsthesia will develop in about ten minutes. In this injection care must be taken not to insert the needle point too close to the point of attachment of the mucous membrane to the bone, otherwise it will be caught up under the periosteum in the canine fossa, when it would be found that further advancement is impossible. A full ten minutes must be allowed to elapse before operation, even though the patient states that a numbness is felt in the cheek. This numbness is due to the anæsthetisation of the terminal branches of the infra-orbital nerve, which, being nearer the surface, becomes affected before the anterior superior nerve is reached. For extraction of these teeth the overlapping branches from the

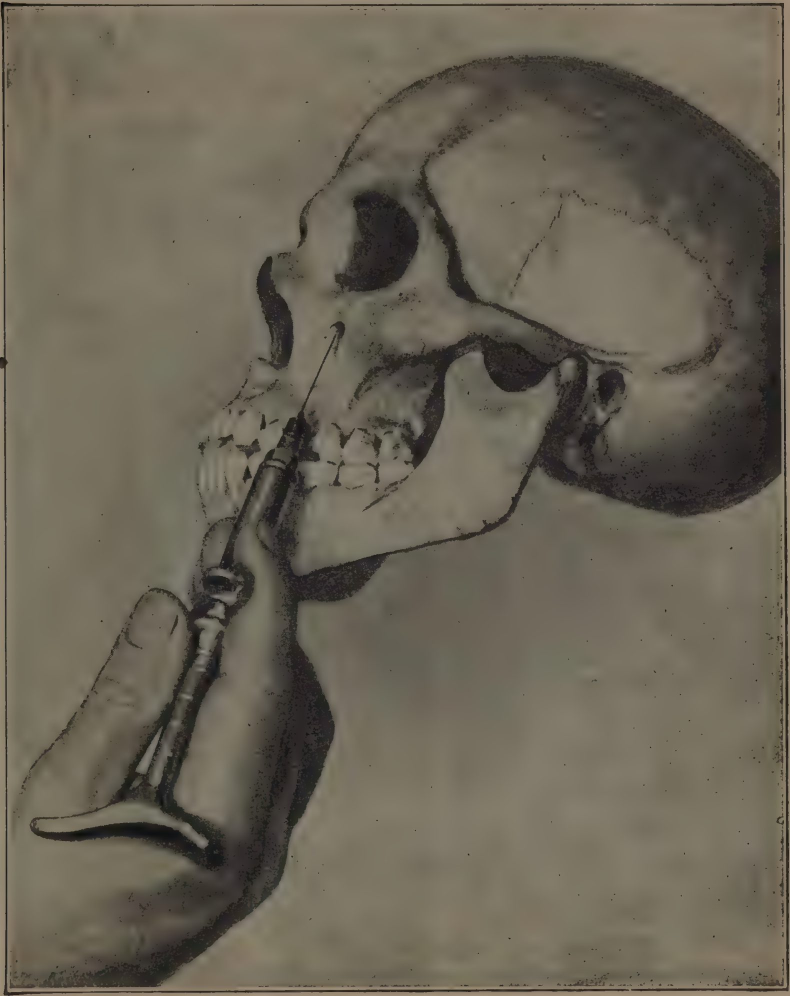


FIG. 973.—The infra-orbital injection.
(From Steadman's 'Local Anæsthesia in Dental Surgery.' Henry Kimpton.)

opposite side must be anæsthetised by a sub-mucous, sub-periosteal injection above the level of the apices of the teeth; an injection must also be given in the palate behind the first incisor teeth, just to one side or other of the mid-line and about half-way between the gum margin and the level of their apices. This injection is painful, but the pain may be overcome by a superficial injection of the gum with half a cubic centimetre of fluid, using a very fine needle a minute or two before making the deeper injection.

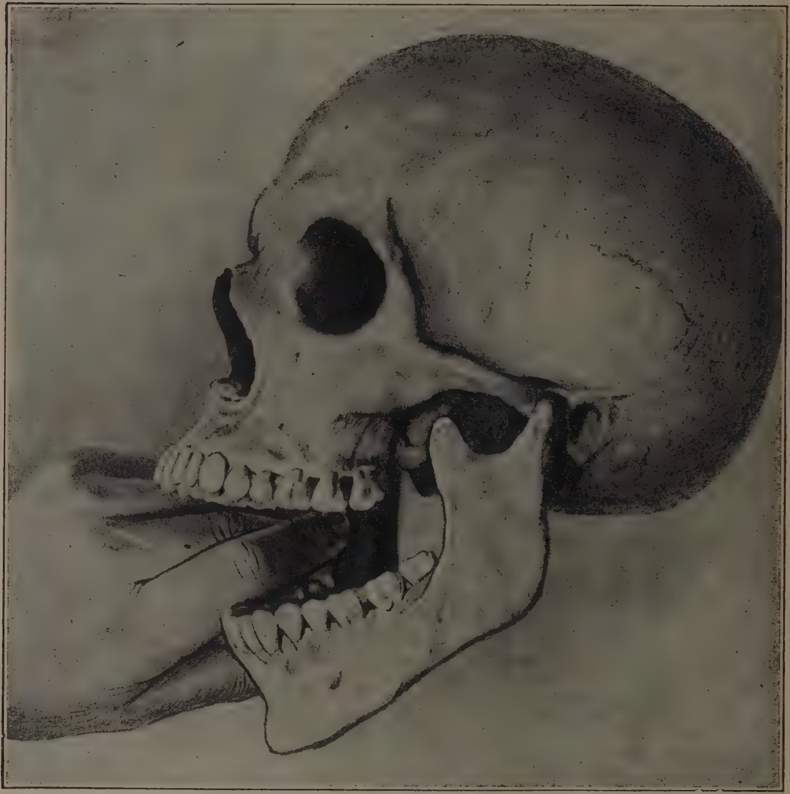


Fig. 974.—Palpating the external oblique line. Second movement. (From Steadman's 'Local Anæsthesia in Dental Surgery.' Henry Kimpton.)

For operations upon the mandibular molar teeth it is necessary to give an inferior dental regional injection. For, owing to the density of both the outer and inner alveolar plates, it is impossible to be sure of obtaining satisfactory results by a sub-mucous infiltration.

There are two methods available for an injection of the inferior dental nerve, the indirect and direct.

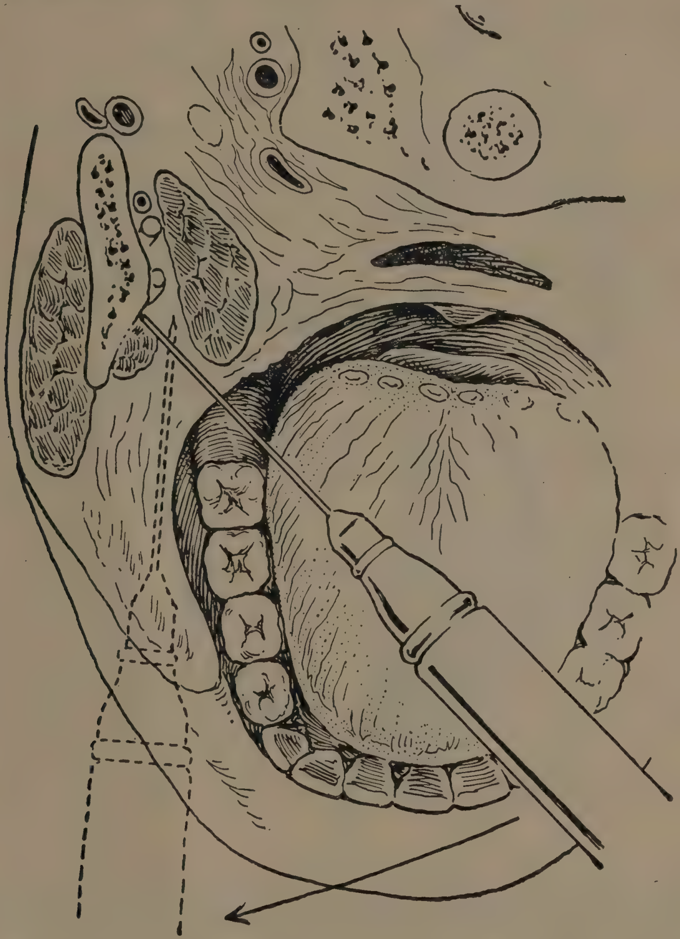


FIG. 975.—Inferior Dental Injection, indirect method : First and second position of needle. (From Steadman's 'Local Anæsthesia in Dental Surgery.' Henry Kimpton.)

Indirect Method.—To give an injection for the right side the operator should stand in front of the patient and place the pulp of the forefinger of his left hand along the external oblique line, and then rotate it inward through a quarter of a circle (fig. 974), taking care to keep the finger parallel with the occlusal plan of the teeth. The finger-tip will now be in the retro-molar fossa, with the nail in contact with the mucous membrane over the internal oblique line. The long straight needle is now inserted into the mucous membrane close to the middle of the operator's nail, with the barrel of the syringe lying in the left corner of the mouth, and is cautiously advanced until the bone is felt at this point. The needle is now slightly withdrawn and the barrel swung over to the opposite angle of the mouth—that is, the right

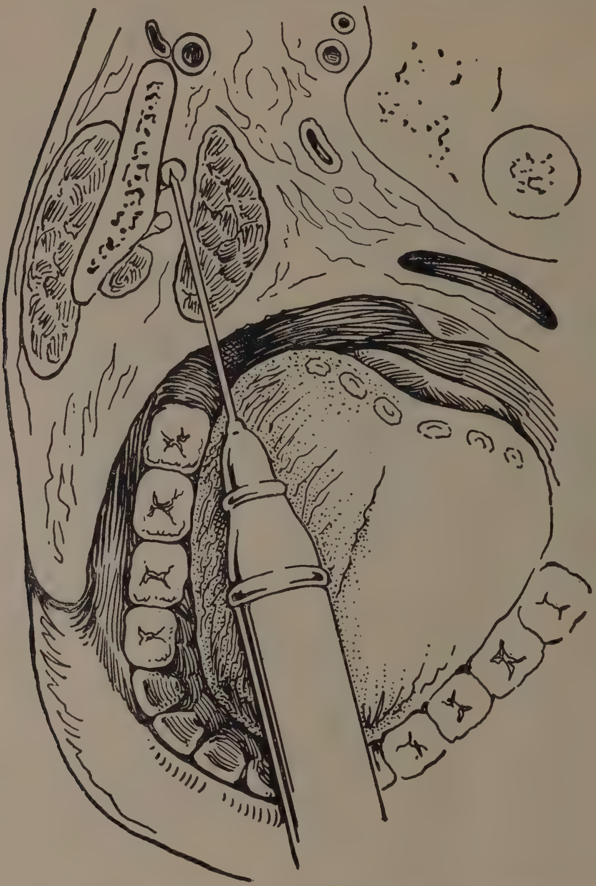


FIG. 976.—Inferior Dental Injection, direct method. Needle passes through the internal pterygoid muscle. (Drawn from a radiogram by Mr. C. H. Howkins. From Steadman's 'Local Anæsthesia in Dental Surgery.' Henry Kimpton.)

—and pushed in a backward direction for about seven or eight millimetres (fig. 975). The barrel is now swung over to the left angle of the mouth, and the needle cautiously advanced until the bone is again felt. The needle point will now be in close proximity to the inferior dental nerve trunk, and the fluid is injected. For the injection on the left side the left forefinger is hooked round the angle of the mouth and placed on the external oblique line. It is then advanced slightly forward until the internal oblique line is felt and the injection is carried out as before.

Direct Method.—The forefinger of the left hand is used as a guide as previously described. The needle is inserted into the mucous membrane about half an inch in front of the finger, with the barrel of the syringe lying over the premolar teeth of the opposite side. The needle is advanced through the internal pterygoid muscle to the bone. This method is rather more difficult to acquire, but its results are certain (fig. 976).

The patient will experience a slight tingling, followed by a numbing sensation at the corner of the mouth about fifteen minutes after the injection. The operation may be safely carried out about five minutes later.

For the extraction of the mandibular second and third molars it is necessary to give an additional injection of half a cubic centimetre for the long buccal nerve. For this operation the needle is inserted midway between the cheek and the gum opposite the second molar. The anæsthesia extends from the third molar to the central incisor of the injected side. If it is required to remove the incisor teeth the overlapping branches of the lingual nerve of the opposite side must be injected by a submucous sub-periosteal injection on the lingual side in the manner described below.

For operations on the mandibular premolar teeth an injection at the sub-mental foramen will suffice. First palpate for the foramen, which is usually to be found below and between the apices of these two teeth. Sometimes, however, it may be found immediately below the apex of the first premolar, or below the apex of the second, and, sometimes, just behind this apex. If difficulty is experienced in finding the foramen it can generally be located by making firm pressure low down in the sulcus. The patient feels a slight pain when the pressure is applied at the point where the mental nerve curls round the sharp anterior border of the foramen. It is important to push the needle point into this foramen. The syringe is held, pen-fashion, parallel to the line of the roots of the teeth and inserted into the mucous membrane two or three millimetres buccally from the junction of the membrane to the bone, and advanced straight downward into the foramen. After injection the fluid should be massaged well into the foramen. If these teeth have to be removed, an injection in the terminal branches of the lingual nerve should be made sub-periosteally below the level of their apices on the lingual side. The double sub-mental injection will anæsthetise the lower incisors and canines, in addition to the first premolars. To anæsthetise the lower incisors and canines an injection may be made in the incisive fossa. The needle should be inserted into the inner side of the lip two or three millimetres from the point of reflection of the mucosa from the bone. Anæsthesia develops in from three to five minutes.

SEQUELÆ.

After-pain.—In a certain number of cases after-pain may occur, varying from a slight amount, lasting for an hour or so, to a severe pain which may last three or four days, or even longer. It is extremely difficult to know how much of this pain may be fairly ascribed to the local anæsthetic. After-pain of very much the same character is not infrequently met with after general anæsthesia and in cases when no local anæsthetic has been given. After-pain is generally due to sepsis. Careful inquiry into cases of pain following upon extraction of teeth seems to show, however, that it is very little, if any, more frequent after local than after general anæsthesia. The chief causes of after-pain are faulty technique, such as, for instance, injections into the muscle or ligaments, scratching the periosteum over the bone, the non-use of an isotonic solution, or the use of a solution too hot or too cold. The administration of too large a dose of suprarenin may lead to excessive contraction of the vessels, and the socket fails to fill with blood. In these cases there is often considerable after-pain, due, probably, to exposure of the nerve endings.

In a certain number of cases, even when every precaution has been taken, after-pain occurs which cannot be traced to any cause.

Treatment.—Massaging the area of operation, together with syringing the socket, often relieves the pain rapidly. A pledget of cotton-wool soaked in dentalone or tincture of guaiacum inserted into the aching socket may bring relief. Failing these, hot dry flannels round the head, the application of the high frequency current, or radiant heat, internal administration of drugs such as aspirin, or a hypodermic injection of morphia in extreme cases may be prescribed.

Toxic Symptoms.—Occasionally toxic symptoms may follow a local injection, which vary from a slight palpitation of the heart coming on two or three hours after the injection, and which is probably due to the suprarenin, to more pronounced symptoms, which may arise soon after the injection. The prominent features are profuse perspiration, dilation of pupils, pallor, throbbing of blood-vessels in the head, laboured and irregular respiration, rapid weak pulse, occasional vomiting, and, sometimes, collapse.

Treatment.—Place the patient in a recumbent position, with the head low, loosen all tight clothing, and direct the patient to take deep breaths ; if necessary, perform artificial respiration. Half to one teaspoonful of sal-volatile in water may be given internally, followed by a cup of hot coffee. In severe cases a cardiac stimulant, such as a hypodermic solution of strychnine sulphate ($\frac{1}{24}$th of a grain) or, if there is respiratory failure, $\frac{1}{120}$th of a grain of atropin sulphate, may be given.

Contra-indications.—Local anæsthetics must not be given indiscriminately. They should be avoided in patients who are in a low state of health, such, for example, as those just recovering from an attack of influenza, and in patients with arteriosclerosis and organic heart disease ; as a general rule, general anæsthesia is safer than local anæsthesia.

CHAPTER XXXVII

Radium and the Dental Surgeon.

By S. G. Allen, M.R.C.S., L.R.C.P., L.D.S.

The science of Radium Therapy is a highly specialised subject, which has developed enormously during the past few years. It has been found that very encouraging results may be obtained in some parts of the body by the use of radium in the treatment of malignant disease. One of these situations is the mouth, and the dental surgeon is frequently called upon to assist the radium therapist. The success of his co-operation will depend largely upon his technical dental skill and ingenuity ; but some knowledge of the properties of the agent used and of the principles of its application is essential. The following is a very brief account of some of the more important facts.

(A) RADIUM AND ITS RAYS.

Radium.—This is an element which differs from most others in that its atoms are in a state of continual disintegration. During this process a definite chain of new elements is produced, each one breaking down into the next of the series, with the liberation of energy in the form of rays. One of the first of the disintegration series is a gas, radon, which may be collected and used instead of the parent radium. It is to some of these rays that radium owes its success as a therapeutic agent. There are three distinct types, known respectively as α, β and γ.

α-Rays.—These rays are positively-charged atoms of helium, which have practically no penetrating power, being absorbed even by a sheet of paper. As radium is always used in some form of container which stops their progress, they do not appear in the field of treatment, and may be disregarded.

β-Rays.—These are negative electrons with greater penetrating power than α-rays. At least 5 mm. of platinum, or its equivalent, is needed to absorb them completely. In their passage through tissue, whether normal or neoplastic, they exert a caustic effect, and, because of the necrosis produced, are only used on special occasions. More often they are filtered off by some form of screen before they reach the area to be treated. β-rays of weak intensity are produced when γ-rays pass through metallic bodies, and this must be remembered when there are fillings or bridges in the mouth or when metallic boxes or screens are used in appliances. It has been found that thin sheets of rubber, vulcanite, or impression material absorb these weak rays. No unprotected metal should be present in the mouth during radium treatment.

γ-**Rays.**—From a therapeutic point of view these are the most important. They are true radiations, similar to light and wireless rays, travelling at the same speed, viz. 186,000 miles per second. They have great powers of penetration. They vary in wave-length, the shortest being the most penetrating, up to 20 cms. of lead being required for complete absorption.

γ-rays produce destruction of tissue also, but in a selective manner. The greatest effect is noticed in tissues which are in an active state of division. Neoplastic tissues come under this category, and, theoretically, by a carefully regulated dose of γ-rays the malignant process may be arrested before the normal structures in the vicinity are harmed. This dose is dependent on many variable and often uncertain factors, and an under- or over-dose may easily be given.

An over-dose of γ-rays, either from excessive treatment or from low tissue-tolerance, results in the so-called ‘radium burn.’ This may be immediate or remote.

Immediate.—Within a few weeks or months of treatment the irradiated tissue breaks down into a painful ulcer. This is usually deeply excavated and lined with tough, green adherent slough. Healing is very slow, and is more delayed in structures with a poor blood supply.

Remote.—This may manifest itself years after treatment in tissues which have been previously subjected to the action of radium, and usually follows an injury, often of only a trivial nature. It probably occurs in situations where an over-dose has not been sufficient to produce immediate necrosis, but has resulted in a progressive endarteritis obliterans of the blood vessels and lymphatics. The tissues are unable to respond normally to trauma, and necrosis results. A slowly progressive ulcer develops, which is limited only by the healthier surrounding tissues. When bone is involved no line of demarcation is formed, and the dead portion remains attached.

In common with other rays, these obey the ‘ law of the inverse square ’—the intensity of radiation is inversely proportional to the square of the distance from its source. This distance is a very important consideration when calculating the dose of radium required, and in the mouth, where space is limited, it is expressed in terms of a few millimetres. It will be seen that any accidental variation by a small amount will greatly affect the calculated dose. Therefore, in the manufacture of radium appliances, as explained later, care must always be taken to measure distances accurately.

(B) METHODS OF APPLICATION IN THE MOUTH.

Irradiation may be carried out :—

(1) Externally :

- (*a*) in applicators containing radium or radon ;
- (*b*) by the radium beam. This is the emanation from a large collection of radium held at distances up to 12 cm. from the skin.

(2) Interstitially : by the implantation into the growth of needles containing radium or radon.

Whichever method is adopted in the treatment of lesions of the mouth, the help of the dental surgeon may be needed. In all cases his first concern will be to produce as healthy a field of operation as is possible under the circumstances. Much can be accomplished in this direction by the careful removal of tartar and debris. Large metal fillings and bridges should be removed, as these produce secondary radiations during exposure.

The question of the extraction of teeth is one on which the dental surgeon must be entirely guided by the therapist with whom he is working. Opinions differ, but it is generally accepted that teeth involved in the growth and those that come into contact with it should be removed. In an unhealthy mouth the remaining teeth might be condemned on general principles, and a clearance advised ; but the wisdom of this procedure depends on the method of treatment to be adopted.

It is a well-known fact that cancer cells may be transplanted into wounds in the neighbourhood of the growth, and one has seen sockets of extracted teeth affected in this way. Where, therefore, purely local treatment in the form of implanted needles or a surface applicator is used, some authorities consider that extractions should be delayed until after the treatment has been given, as the risk of radium necrosis is less than the risk of spreading the growth. Where the radium beam is used, the field of treatment is much wider, and most of the tooth-bearing area may be subjected to its action. This increases the risk of necrosis with subsequent extractions, even in the more remote parts of the mouth. Where extractions are to be performed a short exposure is given to prevent implantation, the teeth are then removed, and after a period of about a fortnight the rest of the treatment is given.

With the radium beam also, one or more of the salivary glands may come within the path of the rays. Their secreting epithelium is affected, with the result that the flow of saliva is appreciably diminished, and the mouth becomes dry. This state of affairs may cause the onset of an attack of very rapid caries, even in previously healthy teeth. Occasionally there is an appearance of general decalcification. Vigorous hygienic measures on the part of the patient and frequent alkaline mouth-washes appear to do little towards the arrest of this condition. It is therefore not at all uncommon for the dental surgeon to be requested to perform total extractions before beam treatment is completed, irrespective of the initial condition of the teeth, especially where the field of application is a wide one, and the prospect of a permanent cure favourable.

(C) RADIUM APPLICATORS.

These are appliances constructed to hold the necessary quantity of radium in a constant, pre-determined position in relation to the growth. They are used in many parts of the body. Where large flat surfaces are to be treated their manufacture is comparatively simple, and is undertaken by radium workers. Applicators for use in the mouth are of a more complicated design, in that they resemble dental plates or splints. For this reason their production is usually entrusted to a dental surgeon.

Impressions of the Mouth.—The first step is to obtain an accurate impression of the mouth, including the affected area. Where the growth is in an inaccessible position it is usual to take a rough impression, and on this to make up a special tray with extensions to carry the impression material. It is often found that, owing to scarring, it is impossible to use as large a tray as one would wish. In these cases the tray should be filled with a large amount of well-softened composition. This is pressed home, and the excess which squeezes out is moulded into position and supported by the fingers until it is ready for removal. There is usually little difficulty in withdrawing such an impression. The use of one of the elastic compositions is advised, as it can be softened readily, it remains plastic for a reasonable time, and when used very soft does not cause great compression of friable tissue. It may be withdrawn before it is absolutely hard, an advantage when dealing with excavated or painful lesions.

Bites.—The bite is taken on wax blocks in the usual way, except that the mouth should be propped open as far as the patient can comfortably tolerate. In this way the maximum space available for the appliance can easily be seen when the models are mounted in the articulator.

(D) MODELLING THE APPLIANCE.

When the models are submitted to the radium therapist he supplies the following information :—

(1) *The extent of the area to be irradiated.*—This is outlined on the model (fig. 977).

(2) *The distance between the radium and the surface of the growth.*—This is a very important factor in treatment. The greater the distance the more even is the field of irradiation. In a cavity such as the mouth one is seriously limited in this direction, and distances of 5 mm. are usual. This is measured from the centre of the tube or needle.

(3) *The distribution of the radium.*—The radium is usually contained in tubes or needles of definite lengths ; less frequently radon is used. It is a great help in the construction of appliances if a number of dummy tubes of corresponding sizes are obtained. These may be handled without danger, and when placed in the required position the form of the finished appliance is more easily

visualised. The therapist arranges the needles according to definite schemes, depending upon the size and situation of the growth, in order to give homogeneous dosage throughout. A random distribution even of the correct amount of radium will produce necrosis.

(4) *The type of filter* (*if any*) *to be employed.*—Where thin-walled tubes or needles are used the therapist sometimes thinks it desirable to insert an additional thickness of metal between them and the growth to act as a filter for the less penetrating and more superficially acting rays. This is usually a sheet of lead or gold which is incorporated in the appliance beneath the needles. The thickness of this is taken into consideration when deciding the ‘ distance.’

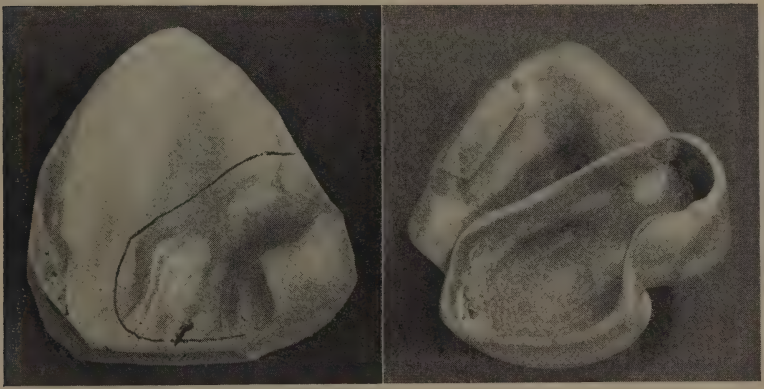


Fig. 977.—Model showing carcinoma of the anterior pillar of the fauces and soft palate, and appliance made to treat this area.

(5) *The form of protection for normal tissues.*—As it is impossible to confine the rays to a particular spot, it is a fundamental principle that the area to be treated should receive the heaviest dose. The surrounding tissues may be protected from excessive radiation by two methods :—

(*a*) By the use of metal screens surrounding the radium. It is found that 1 mm. of lead or its equivalent will absorb about 4 per cent. of the total rays, but a much higher percentage of the soft or more superficially acting rays. In order to diminish the total number of rays appreciably, the lead screen must be of such thickness and consequent weight that its employment in the mouth is not easy. It has been found in practice, however, that a small amount of lead, used in conjunction with other forms of protection, lessens the reaction in normal tissues.

(*b*) Distance. By applying the 'law of inverse square' it will be seen that the further the normal tissues are from the radium the less by a geometrical ratio is the dose of rays received by them. In practice this method is largely used. The appliance is so made that the normal tissues are held back as far as possible from the radium.

(E) CONSTRUCTION OF APPLIANCES.

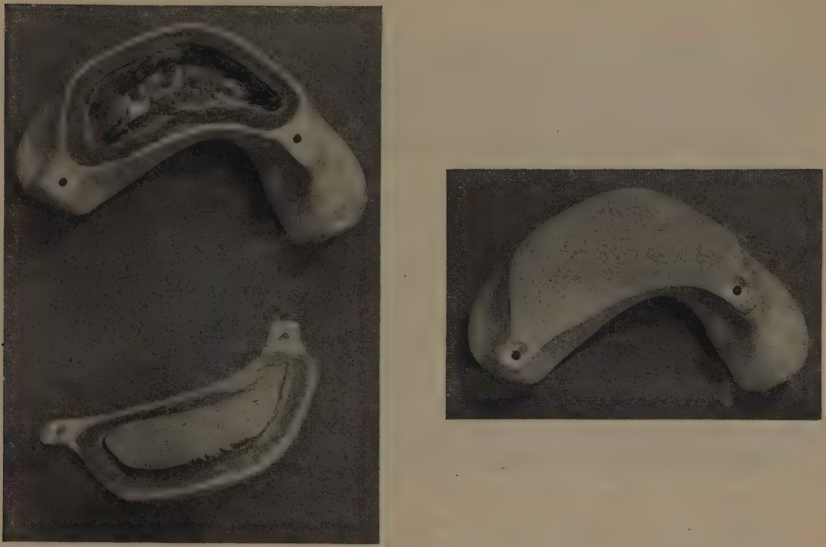
With this information in hand the dental surgeon may turn to the construction of the appliance. This is usually made of :—

(1) Base-plate and impression material. The great advantage of this method is its simplicity. An appliance may be quickly and easily built up with a minimum of apparatus. It is, however, brittle and lacks strength. This is a disadvantage where large areas are to be covered, and where the resistance of fibrous or muscular tissue has to be met, or where there is difficulty in insertion. There is also a probability of disintegration if an appliance of complicated shape is worn for any length of time.

The base-plate is first moulded to the models, covering over the lesion, the alveolar ridges, and the palate, if in the upper jaw. The area to be treated is thickened with composition such as Kerr's or Stent's, to give the necessary distance. Dummy needles are then placed into position. In order to facilitate separation after the next operation, this surface is oiled. It is then covered with another block of warm composition, which is moulded into such a shape that the neighbouring tissues are held away from the needles, and by extensions on both sides the mould is brought into contact with the opposite jaws. This serves to steady the appliance when in use. The whole is now cooled, the two parts separated, and the needles removed. The parts are then reassembled and tried in the mouth, when minor alterations may be made by warming or scraping. The actual needles are then placed in the grooves formed in the second portion, and the two pieces joined together with a hot wax-knife. Grooves for the needles may be cut in the second portion, if dummy needles are not used as described above.

(2) Vulcanite. This has one disadvantage in that the process

of manufacture is longer and more complicated. Skilled assistance in the mechanical laboratory is necessary, and several days may have to be set aside for trying in the case and for finishing. The great bulk of many appliances renders very slow vulcanisation essential if porosity is to be avoided. On the other hand, they are very strong and may be removed and reinserted by the patient without fear of damage, even where there is scarring or muscular resistance to be overcome. Some authorities prefer the use of black vulcanite, as this has a smaller metallic salt content than others, and for this reason less secondary radiation is produced from its surface.



A FIG. 978. B

Radium appliance with cast lead box incorporating a filter at the base, and with a cast lead lid. All external metal surfaces are covered with vulcanite.

The appliance assembled. The lid was held with screws, the heads of which were covered with composition.

When a vulcanite appliance is to be made, a wax model is prepared on the plaster cast. The wax is moulded over the area of the growth to the required thickness. Dummy needles are then placed into the position decided upon, and around them a wall of wax is built, forming a box with its opening away from the growth. Where a filter is to be inserted, a piece of sheet metal (usually lead) of the required thickness is burnished down on the base of the box, being separated from the tissues by the layer of wax. In the final stage this is replaced by vulcanite, which acts as a filter for the secondary radiations from the surface of the lead. In these cases the total thickness of the base—lead and wax—must be measured when calculating the distance of the radium from the growth. The box is now incorporated into a wax plate, which is moulded over the remaining alveolar surface of the jaw concerned.

It is usually more comfortable, and the insertion of a bulky appliance is easier, if a separate bite-plate is made for the retention of the appliance, particularly where there is any limitation of movement of the mandible. This is modelled up on the opposite jaw, with extensions to engage on similar extensions built up on the applicator. These wax models are tried in the mouth, adjusted for bite and fit, and then reproduced in vulcanite in the usual way. The vulcanite appliance is again tried in the mouth and adjusted. The needles are then placed in the box in their original position and held by a little hard wax. If a screen is to be incorporated, a piece of lead sheet of the necessary size and thickness is burnished over these. The remaining space is filled with a mass of warm composition, which is forced into the box holding all securely in place. The walls of the box should be slightly undercut to prevent displacement. When the composition comes into contact with a soft structure such as the cheek or tongue, which can be pushed away easily, it should be built out to give as much protective distance as possible.

Where extra protection is required at the sides of the box, the walls may be cast in lead, together with a filter at the base, if one is to be fitted. Fig. 978 shows an appliance so made. It will be seen that the lead is completely covered with vulcanite to absorb secondary radiations.

Some dental surgeons maintain that the bite-plate as a method of retaining an appliance in its correct position is unsatisfactory and uncomfortable to wear, and claim that, especially in the upper jaw, an external method of fixing should be adopted. This is usually a form of head-cap, with descending rods which connect with the appliance, similar in construction to one form of splint used in the treatment of fractures of the superior maxilla. In one's own experience it has never been found necessary to depart from the use of the bite-plate. If this is made with the articulating rests at the side, keeping the front of the mouth free, speech and respiration through the mouth are not seriously interfered with.

Radium appliances are worn for a definite number of hours, which should be as continuous as possible. They are removed during meals and usually during sleep. The treatment extends over a period varying from several days to several weeks, depending on the dose and the determination of the patient. Owing to their bulk most radium appliances are much more difficult to tolerate than dentures, and for this reason, in spite of their comparatively short period of use, they should be made with every care. The lot of the patient can be made much easier if attention is paid to small details, such as the lubrication of the appliance, to aid insertion, if it is large, the spraying of the throat with cocaine to prevent retching, and the use of a four-tailed bandage to support the lower jaw if the appliance is a heavy one. It is usually remarkable how patients accustom themselves to their presence after a few hours. A loaded radium appliance

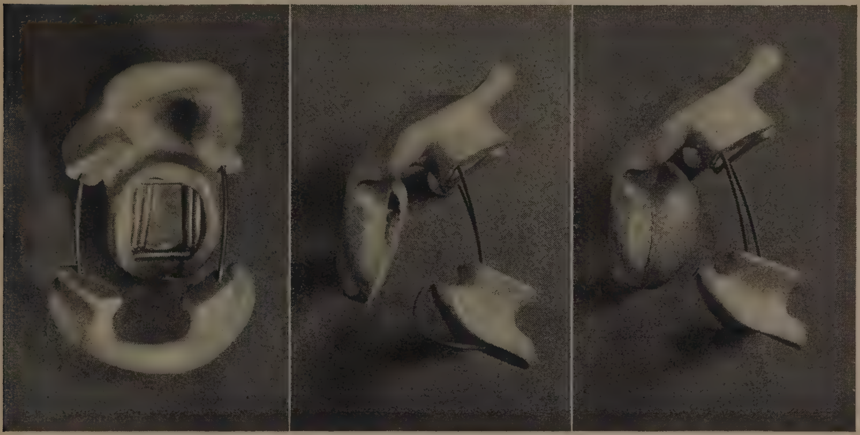

(A) (B) (C)

FIG. 979.—Three views of a radium appliance and bite-plate for the treatment of carcinoma of the soft palate.

(A) Showing the needles *in situ.*
(B) Side view.
(C) Showing composition plug for sealing off the box and for pushing away the tongue.

is a valuable article. When not in use it should be locked away in a special box made of lead to prevent stray radiations.

Something has been said about protection of the patient from excessive radiation. The dental surgeon, who may be brought into contact with radium cases, should remember also to protect himself. Radium needles should only be handled behind suitable lead screens by means of long forceps, preferably non-metallic, to avoid secondary radiations, and loaded radium appliances should only be carried about in special lead boxes. The Radium Protection Committee has laid down the rules for the guidance of radium workers. He should acquaint himself with these, and should avail himself of the means of protection provided at the institution in which he is working.

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